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HEALTH EFFECTS OF AIR POLLUTION

HEARING

BEFORE THE

SUBCOMMITTEE ON
ENVIRONMENTAL PROTECTION

OF THE

COMMITTEE ON
ENVIRONMENT AND PUBLIC WORKS
UNITED STATES SENATE

ONE HUNDRED FIRST CONGRESS

FIRST SESSION

APRIL 18, 1989

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HEALTH EFFECTS OF AIR POLLUTION

TUESDAY, APRIL 18, 1989

U.S. SENATE,
COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS,
SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION,
Washington, DC.

The subcommittee met, pursuant to notice, at 9:58 a.m., in room 216, Hart Senate Office Building, Hon. Max Baucus (chairman of the subcommittee) presiding.

Present: Senators Baucus, Mitchell, Graham, Lieberman, Chafee, Durenberger, and Jeffords.

OPENING STATEMENT OF HON. MAX BAUCUS, U.S. SENATOR FROM THE STATE OF MONTANA

Senator BAUCUS. The subcommittee will come to order. Good morning, ladies and gentlemen.

Today's hearing marks the start of a process that will end with the reauthorization of the Clean Air Act. This hearing will focus on the health effects of air pollution.

Believe it or not, it has been almost 20 years since Congress first enacted the Clean Air Act, and we still have a problem. Our air is so dirty it still threatens our health.

For nearly 12 years we have made several attempts to tighten this law so that we can breathe clean air, but each time special interests have worked against passage. We can not wait any longer to clean up our air.

The situation is very similar to fighting the Federal budget deficit; we can not spend dollars on new programs unless we cut back on existing programs.

We must deal with clean air in much the same way. If we let one polluter off the hook, we must tighten down on another. If the auto industry wants us to loosen standards, we must find greater emissions reductions elsewhere.

For too many years too many polluters have been let off the hook. We have been running an environmental deficit, and we are now paying the price. We still have dirty, unhealthy air and the time has come to pay the piper.

We must make good on a promise that we made to the American public in 1970 when we first passed the Clean Air Act and again in 1977 when we revised the act. Both times, the Congress and the President promised to protect everyone, to keep our Nation safe from hazards of unhealthy air. Unfortunately, we have not kept that promise.

What safety was there in 1985 when almost 77 million Americans lived in areas which exceeded the ozone standard? What safety was there in 1988 when 150 million people lived in areas exceeding the standard? What safety was there last summer when ozone levels in many parts of the country were the worst of the decade? And what safety will there be this year when over half the American population in some 90 areas of the country will breathe unhealthy air—air so contaminated that breathing it will cause some Americans in these areas to get sick and even die? Air so contaminated that it would be illegal to expose a factory worker to it?

Since we revised the Clean Air Act, we have learned that air we once thought was clean may not be so clean after all. The levels we then set to protect us from ozone and other pollutants are now believed to be unsafe. We now have evidence that exposure to ozone at concentrations lower than our current standards causes damage to our lungs. Longer periods of ozone exposure cause even more serious health effects.

We have also learned that carbon monoxide is still a problem in many parts of our country, and this is a cause for concern. Exposure to even low levels of carbon monoxide is harmful. It affects our heart, our sight, our manual dexterity, our learning ability, and may also harm fetuses.

We have also learned that exposure to highly acidic air pollutants like sulfates and fine particles is damaging. These harm our lungs and our ability to breathe. We have learned that in the 12 years since we have revised the Clean Air Act, we have spent millions of dollars in health care costs to diagnose and treat illness and diseases caused by breathing unhealthy air.

These are the reasons to act. We have the remedies we need in hand. We have the Clean Air Act and we have the amendments we will be developing this year. We have existing air pollution technologies as we did in the past, and we will develop even better technologies in the future.

We must act now. We can not wait. Our air is so dirty that a prominent researcher who testified before this committee in 1987 has consistently found that two to five percent of the illnesses and deaths in this country are due to air pollution.

If he is correct, more people may die from air pollution each year than die in automobile accidents. We can not prove this, but the next time you begin to cough or your chest feels tight, maybe you should wonder if the cause is a cold or our dirty air that is causing the discomfort.

Data on hospital admissions of children show that when the air is dirty, more children go to the hospital to be treated for breathing problems. The need to act is compelling.

We inhale about 600 times per hour, 14,400 times per day. This natural life-giving function should not carry with it the seeds of disease and death.

I want to thank all the witnesses for coming here today. I look forward to hearing from them, learning from them, and working with them to achieve a fair and responsible solution to the problem.

I will now turn to my colleague, Senator Chafee from Rhode Island, a very able member of this committee.

OPENING STATEMENT OF HON. JOHN H. CHAFEE, U.S. SENATOR FROM THE STATE OF RHODE ISLAND

Senator CHAFEE. Thank you very much, Mr. Chairman.

First of all, I want to congratulate you for making health effects the topic of our first hearing on the Clean Air Act Amendments this year. This testimony is going to set the stage for the coming months of work on the clean air legislation.

I hope everybody was here when the Majority Leader said that clean air legislation will be considered in this Congress. That is a very significant step forward from previous years, when we reported bills out from this committee without having them considered on the Floor. All that has changed with the very flat statement from the Majority Leader. That is an echo of what he has said previously on the same subject.

This is the beginning, Mr. Chairman, of many hearings which we are going to be having in this committee under your fine leadership over the next two months. This morning we submitted the first of three parts of legislation on clean air—that is, on toxic air pollution. The acid rain provision and the bills to deal with nonattainment are yet to come, namely dealing with ozone and carbon monoxide.

The protection of public health is a primary goal of each of these bills. That is what we are dealing with, trying to protect the public health. The hearing today will identify some of the subtle and some of the not so subtle effects on health that we need to address.

Asthma attacks from sulfur dioxide certainly are not subtle. Ozone levels that limit the amount of exercise that healthy adults can undertake are not subtle.

We are not primarily talking about body-count when we discuss dirty air, although air pollution almost certainly contributes to additional deaths among our elderly and those who are especially vulnerable. However, we are talking about some real economic effects of unhealthy air. More important, we are talking about serious and widespread erosion in what we call the quality of life.

I look forward to hearing the witnesses today, Mr. Chairman, and congratulate you for assembling them. Thank you.

Senator BAUCUS. Thank you, Senator.

Senator Durenberger.

Senator DURENBERGER. Mr. Chairman, I have no opening statement, thank you.

Senator CHAFEE. Mr. Chairman, I have a statement from Senator Warner that I would like to submit for the record at this time. Also, Senator Jeffords had to leave and would like his statement entered into the record.

Senator BAUCUS. Fine.

[The statements referred to follows:]

OPENING STATEMENT OF HON. JOHN W. WARNER, U.S. SENATOR FROM THE COMMONWEALTH OF VIRGINIA

Thank you, Mr. Chairman.

Clean air legislation involves many complicated and technical issues. As this Subcommittee considers clean air legislation, it is important that we not only understand what such legislation achieves as a technical matter, but why we are enacting it and its broader implications. It is critical that in making difficult decisions about

clean air legislation, this Committee fully understand all the ramifications of such legislation.

I hope that this is the first of a series of hearings that will consider the underlying justification for and effects of clean air legislation. I hope, Mr. Chairman, that the Committee will also consider the impact of legislation on various regions of the country, the positive and negative effects on the quality of life for our citizens, and the effect of such legislation on our Nation's economic and competitive position in the world. I look forward to further Subcommittee hearings on these and other matters.

But the health of our people must be our first and foremost concern. I consider my service on this subcommittee a very important part of my service to the people of Virginia. The Environmental Protection Agency recently released data showing that Virginia ranked fourth in the Nation in the amount of toxic air pollutants. It is particularly appropriate, therefore, that we hold this hearing exploring the health effects of air pollution. For that reason, Mr. Chairman, I wish to commend you for holding this hearing today.

I want to welcome the witnesses and I look forward to hearing their testimony.

OPENING STATEMENT OF HON. JAMES M. JEFFORDS, U.S. SENATOR FROM THE STATE OF VERMONT

Thank you, Mr. Chairman for convening this hearing today to hear testimony on the health effects of air pollution. We have a very distinguished group of witnesses present, and I look forward to the discussion.

Many in my home state of Vermont feel that we already have adequate information to justify regulations that would reduce the amounts of pollutants that are daily spewed into our atmosphere. It is no mystery that carbon monoxide can cause death at high doses—today we should learn more about its effects at lesser doses. As for ozone, my instincts tell me that smog is less healthy than fresh air, but today's hearing should reconfirm this. And in regard to sulfur dioxide, nitrogen oxides and other precursors of acid rain, I have long held that the environmental damages being caused by these pollutants justify the costs of controls. It disturbs me that many still deny the problem even as we compile more evidence of health effects of acid rain.

We in the Congress are charged with formulating decisions based on a strong showing of evidence. Indeed, for some it seems that irrefutable proof that the benefits of an action outweigh the costs of providing that benefit is a necessary prerequisite for taking that action.

I reject the necessity of such strict criteria. Scientists, lawyers and legislators can argue themselves to the grave before absolute, conclusive evidence has accumulated in the laboratory. The scores of dead lakes in the Adirondack mountains of New York, and the stands of Pine atop Camels Hump in Vermont and Mount Mitchell in North Carolina bear credible testimony to the destructive effects of air pollution. Ozone incidents at Acadia National Park in Maine, and last year's air here in Washington, when the weather forecasters warned us to keep our children indoors, is further evidence that air pollution needs curtailment.

But in this business we must make decisions based on showings of fact. We must cut through emotions and instincts to target the most cost-effective and necessary actions. In times of budget deficits and international competitiveness, we must strive to get "the biggest bang for the buck."

Today's hearing seeks to quantify some to the things that some deny as strongly as others support. Today's hearing will contemplate scientific studies and epidemiologic data. Today we entertain the knowledge of some of the brightest professions on the subject of health effects of certain pollutants.

Mr. Chairman, I look forward to a productive hearing.

OPENING STATEMENT OF HON. BOB GRAHAM, U.S. SENATOR FROM THE STATE OF FLORIDA

Senator BAUCUS. Senator Graham.

Senator GRAHAM. Mr. Chairman, I ask unanimous consent that my statement be included in the record.

Senator BAUCUS. Without objection, so ordered.

[Senator Graham's statement follows:]

OPENING STATEMENT OF HON. BOB GRAHAM, U.S. SENATOR FROM THE STATE OF FLORIDA

Mr. Chairman, I appreciate the opportunity to hear from an array of witnesses on the health effects of air pollution, particularly those involved n acid deposition and mobile source emissions.

Legislation, of which I am proud to be a cosponsor, is being introduced today to better regulate emissions of air toxics, and we are in the midst of drafting new clean air legislation to address both the acid rain and ozone and carbon monoxide nonattainment problems.

As those of you who have been here throughout the many years of discussion are keenly aware, this has been a long and arduous process. We will hear testimony today that emphasizes that we do not have the luxury of waiting any longer. This Congress must be the one in which we accomplish our goal of reauthorizing the Clean Air Act.

While Florida does not experience the magnitude of problems that some other States do in this regard, we have a large population of elderly citizens who exhibit greater sensitivity to these pollutants. For those areas in Florida out of attainment for ozone alone, there are an estimated 1.2 million elderly citizens—those over 65 years of age—out of a total elderly population of 2.3 million (greater than half).

These figures do not take into account the volume of people moving into the State of Florida daily—estimated at 1,000. Children under the age of 13, who are also very susceptible to the adverse effects of ozone and other criteria pollutants, comprise 12.3 million of those residing in Florida's ozone nonattainment areas.

In addition to these residents who are vulnerable due to age, many other citizens are sensitive due to existing respiratory illnesses. I represent a State which attracts people with its warmth and sunshine—both of which serve as catalysts in converting hydrocarbons and nitrogen oxides to ozone. We aren't going to change the warmth and sunshine; we must therefore concentrate on the sources of emissions that react to form ozone.

Mr. Chairman, I look forward to hearing from our expert witnesses today and hope that their testimony can serve to aid in our expedited passage of a reauthorized Clean Air Act. Thank you.

Senator BAUCUS. Before beginning with the witnesses, I would like to announce a rearrangement of the panels due to time constraints and to schedules to accommodate various witnesses. Panel Three will come second. Mr. Walker will then be first on the second panel, and Dr. Green will be first on the third panel.

We will begin now with Panel One, Dr. Bromberg and Dr. Lippman, if you gentlemen will please come forward.

You will have ten minutes each, gentlemen.

Dr. Bromberg, please proceed.

STATEMENT OF PHILIP A. BROMBERG, UNIVERSITY OF NORTH CAROLINA, CHAPEL HILL, NC

Dr. BROMBERG. Thank you, Mr. Chairman and Members of the Committee.

I am Philip Bromberg, a specialist in lung diseases and internal medicine, professor of medicine at the University of North Carolina School of Medicine and also director at the same institution of the Center for Environmental Medicine and Lung Biology. I am pleased to have the opportunity not only to discuss health effects of ozone and carbon monoxide, which continue to be two of the air pollutants of considerable concern, but I also have been requested by the committee to introduce the general subject of health effects and the way in which air pollutants act directly upon the lung or are absorbed through the lung to act upon the other tissues.

It may seem difficult to imagine that ozone concentrations of as little as a tenth of a part per million, that is, there are only 100 molecules of ozone among every billion air molecules, could cause

any health problems at all, although those of us with allergies know that inhaling minuscule amounts of pollen, animal dander, or house dust can wreak havoc in the airways and the lungs.

Before talking about ozone or carbon monoxide, I would like to simply show a few diagrams of the area of the body that we are talking about. In this schematic cutaway of the upper part of the human body, on top toward the right, you see the nasal cavity with its surface lining, the mucosa, and you see the oral cavity. These join at the pharynx, which then leads down to the windpipe (the trachea), which then branches progressively into smaller and smaller conducting airways, and finally into sacs shown on the bottom right, which are called alveoli.

The alveoli is where the primary function of the lung is conducted. That is, the uptake of oxygen into and the excretion of carbon dioxide from the blood. Those air sacs are very thin-walled structures in which the separation between the air and blood is perhaps one one-hundred-thousandth of an inch. The blood flows through the capillaries in a more-or-less continuous manner while the air moves in and out so that with each inspiration you bring some fresh air into the alveoli and with each expiration you exhale whatever has been excreted from the blood into the alveolar air. The surface available for gas exchange in the adult lung is about the size of a tennis court. This makes the lung a highly effective site for rapid uptake into the blood of inhaled vapors. "Crack" users take advantage of this fact.

On the other hand, the airways do not participate in the process of oxygen and carbon dioxide exchange. They serve as conduits and are relatively thick-walled structures. They are lined by a layer of epithelium. In their will they contain circularly-arranged smooth muscle that can constrict to reduce airways caliber, glands that can secrete substantial amounts of mucus, and nerves and mast cells that can serve as amplifiers so that a small stimulus may be translated into a large response.

Thus the two functional areas of the lung are: the conducting airways and the gas exchange area. Toxicants that affect the airways are going to cause symptoms like cough, sputum production, wheezing, tightness in the chest, and perhaps a feeling of irritation or pain underneath the breastbone. Toxicants that affect the alveolar, the gas-exchange portion of the lung, are more likely to produce in the long run, diseases like emphysema, scarring of the lung, or an exuberant production of collagen fibrils that stiffens the lung and prevents it from expanding easily in a homogeneous manner. More acute alveolar injury will cause gross leakage of fluid into the air spaces as is seen with exposure to phosgene or to very high levels of ozone.

Some air pollutants will cause damage neither to the airways nor to the alveoli. This is the case for carbon monoxide and for a number of organic chemical vapors. For these substances the lung serves as a portal of entry into the bloodstream. Further transport of the agent to sensitive tissues occurs via the blood. Health effects of such agents involve organs other than the lung. I will now return to the lung as a target organ.

The next schematic is of the surface lining layer of the airways. I emphasize the surface because this is the contact point between re-

active pollutant gases like ozone (and other toxic gases) and the respiratory tract. Below you see epithelial cells lined up side by side, represented as columnar cells with a big nucleus. We see that most of these cells have on their surface hair-like appendages in large numbers called cilia. These are continually beating in such a manner as to convey particles trapped on the mucus layer toward the larynx and then the pharynx where they are swallowed. We have an important clearance mechanism here, very ingeniously arranged, that keeps the airways surface free from inhaled particles and helps maintain their sterility.

Also on the left of the slide, you can see that one of the cells is not a hair-bearing or ciliated cell. It is a mucus-producing cell, and those gray globules are mucus that is being discharged on to the surface and is helping to create that sticky surface layer.

Not shown on this side is the rich nerve supply that courses between and among these surface cells as well as among the deeper tissues of the airway wall. Just below that cell layer are other potent effector cells such as mast cells which are so important in allergy. The reason I emphasize these elements is that small insults can produce large effects because the neural network and the mast cells constitute amplification mechanisms.

These mechanisms not only stimulate airway smooth muscle to constrict and glands to secrete, but they cause liberation of attractants that call in other cells (inflammatory cells) to complement and prolong the overall response to inhalation of a toxicant.

Now I would like to show you a kind of topographic view of the surface epithelium. You can see that most of the surface is covered by ciliated cells, but in between there are white globes representing mucus-producing cells. If we look (at much higher magnification) at cross-sections of these cells, we see (above) the cross-section of one of these hairs—each one of those complex little dark circles is the cross-section of a single cilium. You can see that they are all separate from one another.

Below you see what happens in the nasal mucosa of a normal human subject after the inhalation of 0.75 parts per million of sulfur dioxide for a two-hour period. Before exposure, all of these cilia are separate. Afterward, the cilia have been agglomerated into a compound structure. When we section the epithelium at right angles to the previous picture we again see this abnormal encapsulation of multiple cilia into a single compound structure.

On the next slide, I want to show similar changes that have been observed recently by members of our group in the nasal mucosa of young children who are living in households of cigarette smokers. Although these data are still preliminary, they again suggest the power of air pollutants to cause changes in the surface cells, if you look for them in the right manner. Most of the nasal mucosal cells are normally ciliated and only a few are mucin-producing. But in the biopsy we have been examining, we see just the opposite. There are many mucin-producing cells and very few ciliated cells. Such changes must adversely affect mucociliary clearance.

I will forego oral comments on ozone and carbon monoxide that are to be found in my written testimony, and I am sure Dr. Lippman will deal with those air pollutants. Thank you.

Senator BAUCUS. Thank you very much, Doctor.

Dr. Lippmann.

STATEMENT OF MORTON LIPPMANN, NEW YORK UNIVERSITY MEDICAL CENTER, TUXEDO, NY

Mr. LIPPMANN. Thank you very much, Mr. Chairman.

I am a Professor of Environmental Medicine at New York University Medical Center, and I very much appreciate your invitation to testify here today on the health effects of air pollution.

I have personally been conducting laboratory research on such health effects since 1975. Since 1980, I have been conducting a series of studies in the field, looking at directly measurable effects of air pollution in natural settings.

I have also participated in the Clean Air Scientific Advisory Committee, generally known as CASAC, in their reviews on the EPA criteria documents and staff papers since 1980, looking at all the criteria pollutants. I was Chairman of CASAC between 1983 and 1987.

I also recently prepared an extensive review of the health effects of the six criteria pollutants for the Congressional Research Service, and the committee staff has made copies of this document available to members of the subcommittee.

Based on this review, my research, and my CASAC experience, I believe that ozone and acidic aerosols are currently having by far the greatest impact on public health of any of the ambient air pollutants. Both are secondary pollutants, that is pollutants formed within the atmosphere from gaseous precursors. Thus, their concentrations are elevated over extremely broad regions of the country and exposures are often higher and the daily peaks more prolonged in regions many miles downwind of major population centers than they are within the centers themselves.

Within the cities, nitric oxide from motor vehicles scavenges ground level ozone effectively, and ammonia from biological decay neutralizes acidic aerosols. Thus, urban pollution monitors that we depend on for our indications of exposure may underestimate regional average exposures. Also, the control strategies based upon local source inventories may continue to be as unrealistic and ineffective as they have proven to be for ozone in recent decades.

In the limited time available this morning, I will emphasize human exposures to, and the health effects of, ozone. Other pollutants are covered in my written testimony.

We know that controlled human inhalation exposure studies in chambers have demonstrated that acute effects of inhaled ozone include decrements in lung function, increases in symptoms, increases in reactivity to other irritants and antigens, increased membrane permeability, depressed particle clearance from the lungs, and an influx of cells associated with lung inflammation. Of particular note, many of these studies have been conducted at levels of exposure comparable to those occurring frequently in ambient air.

At any given ozone concentration, the severity of the response increases directly with the duration of exposure and the volume of air inhaled. Thus, the greatest responses to ozone occur among

people who spend many hours out of doors engaged in vigorous work or play.

Working groups at risk include construction workers, farm workers, postal workers, police officers, etc. Other groups at risk include those engaged in strenuous recreation, including adult joggers and tennis players, as well of course, as children of all ages engaged in vigorous play out of doors.

Until now most environmental health scientists have relied upon the exposure response data from controlled exposures in chambers where the ozone is present in otherwise purified air. In one very important test recently performed at EPA's laboratory, 22 healthy young men were exposed to .08 parts per million of ozone for 6.6 hours, including 5 hours of moderate exercise.

Respiratory function as indicated by the forced expiratory volume in one second, decreased with each succeeding hour of exposure. At the end of the study, it was reduced by an average of 7 percent, with one individual having a 26-percent reduction. Greater responses were produced at higher concentrations.

EPA has proposed and the CASAC Committee has agreed, that the effect becomes adverse when the magnitude of the change exceeds 10 percent in an individual. In the .08 study, seven of the 22 individuals crossed that threshold. The 6.6 hour exposure studies clearly demonstrate that the 1-hour averaging time in the current ambient air quality standard is inappropriate and should be replaced with a 6-to-8 hour averaging time in future standards.

While studies in chambers provide the most precise data, they do not adequately represent the effects that occur in more natural settings. In a study that I directed involving 30 young adults who exercised outdoors at our Tuxedo, New York laboratory in relatively clean air for a half-hour each day during four weeks of the summer of 1985, the FEV1 decrements associated with ambient ozone were almost as large after half an hour as those seen after 6 hours in the chamber exposures.

Thus, the effects were accumulating at a much greater rate than in the chamber. Similarly, the effects we have seen in our 1980, 1982, 1984, and 1988 studies of children in summer camps have been much greater than those seen in children exposed to ozone in purified air in the chambers. The potentiation of the characteristic ozone response in these studies is most likely due to acid aerosols in my view, but further research is needed to confirm this hypothesis.

I have been talking about the acute effects of ozone. They are largely, but not entirely, reversible within one day, and the contributions of these acute effects to chronic lung disease are not yet known. There is good evidence that repetitive ozone exposures at levels comparable to those frequently encountered in ambient air do produce chronic lung damage in rats and monkeys. Their lungs become less elastic and the lung capacity is reduced.

There has been one human population study in Southern California suggesting a loss of lung elasticity over a 5-year span with the higher ozone community having substantially more loss than the lower ozone community. The low ozone community in California has more ozone exposure than most communities in the rest of the country, and the rate of loss in that lower ozone community was

abnormally high compared to published literature on comparable populations elsewhere.

Considering that millions of people are being chronically exposed to high levels of ozone, serious concern about accelerated lung aging is warranted. Additional research focused on the chronic effects of ozone is clearly of high priority.

There is a general perception that ozone is present at concentrations of concern only in California, but this is clearly not the case. As the Chairman mentioned earlier, half the population or more is exceeding the current standard.

Figure 4 of my testimony shows that the maximum daily 8-hour average is usually at least 90 percent of the maximum hour average. While 120 parts per billion for 1 hour might not produce a large response, people can be outdoors for these longer exposure periods and the effects accumulate rapidly.

As I have indicated, one reason is that ozone is a regional pollutant. The widespread ozone contamination increases both the number of people exposed and the magnitude of their exposure in warm weather when the largest number of people are outdoors for extended periods. Similar considerations apply to acidic aerosols.

I would like to indicate that there has been a lot of progress in the research field on these acute effects of ozone. We now understand them pretty well. On the other hand, we know hardly anything about the chronic effects of ozone, which may produce an accelerated lung aging analogous to that produced by cigarette smoke exposure.

I think that while we need research on the chronic effects of ozone, we need to take action to reduce exposures as much as possible in the near future. During this period when interim steps are being taken to reduce exposure to ozone and acidic aerosols, and their effects, EPA should be given a mandate and the means to conduct and support the research needed to design and implement the long-term prevention and health protection goals of the Clean Air Act.

EPA has undertaken some valuable research, and the National Institute of Environmental Health Sciences [NIEHS] has also been quite active, too. NIEHS and EPA were joint sponsors for a symposium on the health effects of acidic aerosols held recently. For the most part, however, NIEHS does not support exposure response-type studies which we need for standard setting. That job belongs to EPA.

Congress needs to recognize that EPA needs the authority and the funding to address longer-term research goals. It needs to be able to commit to programs that will take up to five years or more to complete, and to protect the funds for these programs from diversion to the short-term regulatory agenda which now drives the EPA research program almost exclusively.

This is a real problem for the agency and for the public. If we are going to address the chronic effects, which are perhaps the most serious effects, then we need to initiate studies now and take this long-term perspective. Exposures are bad now, but they are not going to get better for ozone in the foreseeable future. We really have to pin down the effects of chronic exposure.

Thank you, Mr. Chairman.

Senator BAUCUS. Thank you, gentlemen, for that excellent testimony.

We will hear probably later today and certainly in subsequent days from those who are opposed to Congress taking forthright action, that the adverse effects of some air pollutants, maybe including ozone, are temporary. That is, that they are easily and quickly reversible. I wonder if you could address that assertion, either or both of you.

You have talked about the chronic versus the acute effects, but I wonder if you could more specifically focus in on the assertion that we will hear, not only today but in subsequent days, that these effects are reversible and, therefore, the Act should not be as concerned about this as perhaps earlier evidence might warrant.

Dr. BROMBERG. I would make three points about the reversibility of ozone-induced effects. First of all, although certain acute effects such as pain in the chest, inability to take a deep breath, or cough may regress within hours after cessation of the exposure, other ways of testing the respiratory system demonstrate effects of a single ozone exposure that last for at least 24 hours.

Secondly, we now know that the exposure induces not only irritative symptoms and inability to take a deep breath, but causes an inflammatory reaction as well. That is an injury amplification of the type described earlier, where inflammatory (pus) cells are actually exuded on to the airway surface. This can be demonstrated in human subjects by performing a procedure called broncho-alveolar lavage. In the salt water that is used to wash out the lung, you can demonstrate a greatly increased number of inflammatory cells many hours after termination of the ozone exposure.

Inflammation is not a desirable process in the lungs. Inflammatory cells incorporate powerful destructive mechanisms that can cause tissue damage and destruction and thus may enhance the development of chronic changes.

Thirdly, there is evidence from longer-term studies of health in Southern Ontario and in the Los Angeles area and from studies of panels of asthmatics in Los Angeles and Houston, that the incidence of adverse respiratory health effects is related to the fluctuations of levels of oxidant air pollution (and perhaps associated materials like acid sulfates). Without reference to animal studies, therefore, and focusing entirely on human studies, I think there is enough evidence that ozone exposures have the potential to cause long-term deleterious effects, that it is prudent public health policy to do all we can to reduce the levels of ozone in ambient air.

Senator BAUCUS. Dr. Lippmann, do you have anything to add?

Dr. LIPPMANN. Yes. I think Dr. Bromberg has covered some of the imporant considerations very well. Certainly now that we know that it is not just a functional deficit, which was focused on for years, but that inflammatory cells are recruited into the lungs, we now have to be much more concerned, as he said.

We also know that in animal studies which have been done at EPA recently, the adaptation of function does not preclude the progression of the damage in the airways.

Senator BAUCUS. Could you explain that point a little more clearly, please?

Dr. LIPPMANN. If you expose humans in a chamber on day one, you get perhaps a 15-percent decrement. If you expose them on day two, you may get an 18-percent decrement. On day three, 10 percent. By day five, hardly any. It appears that if you were exposed every day, the effect goes away over a period of some days.

This has been reproduced in a rat model. It is clear in the rat model that while the function adapts, the underlying damage of the kind that Dr. Bromberg is describing continues to progress even though the function, the ability to take a full breath, has been restored.

It is also of interest that it takes about three times as much ozone to get the same response in the rat as you get in the human. Other work on dosimetry indicates that two to three times as much ozone penetrates to the active area in the deep lung as in the human, making it consistent that there be this lesser response among rats.

Senator BAUCUS. What happens if it is intermittent?

Dr. LIPPMANN. That can be worse. In fact, the evidence is beginning to accumulate proving that intermittent exposure is worse than constant exposure. I refer to a study performed on monkeys at the University of California, Davis, where animals were exposed 8 hours per day, 5 days per week, for 18 months.

Half the animals were exposed every day on a 5 day per week schedule and an equal number of monkeys were exposed every other month, 5 days per week for 4 weeks, 4 weeks off, and then repeated. The effects were as large and in many end points larger in the animals getting half as much ozone, corresponding to a series of seasons.

The reason, in simple terms, is that some of these chronic diseases are the result of ineffective defense of the body. The body gets geared up to the challenge, gets turned on and turned off, and the natural defense cells in the lung responsible for maintaining homeostasis secrete enzymes which contribute to disease.

I think we have two examples here of the drunk under the lamp post syndrome. The toxicologist is at fault for always trying to look for an effect with a maximum tolerated dose. This precludes the kind of chronic effects that we see at lower doses. When the toxicologists get more sophisticated and do more realistic patterns of exposure, they can see greater effects.

Likewise, the lung physiologist knows how to make function measurements very exquisitely, and for years all they did was measure function. It is only recently that they had a little bit more imagination to wash the lung and find these inflammatory cells.

The repeated damage is the reason for our concern. And the animal models, while not exactly like humans, give us no comfort. They give us concern.

Senator BAUCUS. Thank you.

Senator Chafee.

Senator CHAFEE. Dr. Lippmann, we all know about the conditions in Los Angeles where the ozone levels are veyr high and so are the carbon monoxide levels. I must say that I am not sure about acidic aerosols. What are they, basically sulfur, or what is in them?

Dr. LIPPMANN. Again, it is a failure of imagination. People have measured particle mass or sulfate ion because that was easy to

measure. It is now quite clear that the active agent in the particles which was producing the mortality and morbidity effects associated with the particles was the hydrogen ion, the free acid formed when sulfur dioxide becomes sulfuric acid as a secondary pollutant.

Senator CHAFEE. So it basically stems from sulfur dioxide?

Dr. LIPPMANN. Sulfur dioxide is the precursor, not the agent itself.

Senator CHAFEE. I see. How come anybody is alive in Los Angeles after all this?

Dr. LIPPMANN. There is probably some long-term adaptation.

Senator CHAFEE. Are there any studies that show that the hospitalization is greater in Los Angeles or that young children are affected? There must be some studies that have been done in Los Angeles, aren't there?

Dr. BROMBERG. There is a study that looked at panels of asthmatics living in the Los Angeles area which demonstrated correlation between changes in air pollution levels and changes in the symotins and appearance in medical facilities of these patients. Then there is a long-term study by a group of investigators that has examined the difference in the rate of decline of lung function with age of people who are residents of a relatively low pollution area of the Los Angeles Basin and those residing in a relatively high oxidant pollution area. This study suggests that those who live in the high pollution area have an accelerated loss of lung function with time as compared to those living in the low pollution area.

In Southern Ontario, with its nationalized health system, Dr. Bates and his colleagues have looked at the correlation of changes in air quality monitored all across Southern Ontario with the health records of the 6 million people who live there. They find, summer after summer, that the incidence of respiratory diseases correlates with the fluctuations in oxidant and acid sulfate air pollution in those areas.

Senator CHAFEE. Dr. Lippmann, Dr. Green's testimony, which we are going to have following yours, addresses the summer camp studies of children. He states,

> Citing statistical differences in such subtle changes of physiological functions without noting the clinical significance can create a false impression regarding the importance of the finding.

What do you believe is the significance of these summer camp studies, and could you comment on what you believe we should regard as adverse health effects? First of all, what do you think of those summer camp studies?

Dr. LIPPMANN. Obviously, I am going to think highly of them since I directed all of them.

Senator CHAFEE. Well, that's a good reason to be for them.

Dr. LIPPMANN. And of course, when doing studies in natural settings, one has to look for what one can find. Clearly, we are not able to do lung washing in the camp setting or some of the other tests which might be possible and desirable in a laboratory setting.

Still, if you want to accept 10 percent change in these basic respiratory functions as a limit—we had children on days that were within the standard, who exceeded that kind of magnitude of a

functional change in the camp. You do what you can under the circumstances.

I might point out that I mentioned in my testimony the study that Dr. Bromberg just referred to in Southern California, talking about function losses which are normal with aging at about 30 milliliters per year in Tucson where there is an occasional exceedance of the standard, perhaps once each year. In the low oxidant community in Southern California where they had 58 exceedances per year, they were losing about 45 milliliters of lung capacity per year. In their high community where the standards were exceeded in those years on 117 days per year on average—twice as many—they had about 60 milliliters per year in loss of function.

Senator BAUCUS. What percent of lung capacity is that?

Dr. LIPPMANN. This would be analogous to what you see in smokers. It accumulates very slowly, and it took us decades to realize the chronic effects of smoking. I am not talking about cancer now, but about emphysema and loss of lung capacity. These things have been going on for some time. The harvest may be yet to come.

Dr. BROMBERG. One percent per year, but in 10 years, that is 10 percent. If you double that, then it is two percent per year. In 10 years it is 20 percent.

Dr. LIPPMANN. Those are baseline changes, not transient changes which are largely reversible.

Dr. BROMBERG. One might just think about cigarette smoking for a moment. The effects of smoking a cigarette are really quite mild if you measure lung function with the same type of test that has been used for ozone studies, a reversible, mild broncho-constriction and some cough. Yet, we now know that aside from the risk of lung cancer and other cancers, there is clearly a strong association between repeated inhalation of cigarette smoke and the development of obstructive lung disease.

Indeed, one of the problems that epidemiologists encounter in identifying the various risk factors for chronic lung disease, is that there is a tremendous baseline effect of cigarette smoking. Here, then, is an example of an apparently innocuous material whose inhalation causes transient reversible effects that in the long term has turned out to be pretty deadly.

Senator CHAFEE. Thank you, Mr. Chairman.

Senator BAUCUS. Gentlemen, I wondered if you could tell me basically what you think an adequate margine of safety would be for ozone. As you know, in the current law it is about 0.12 parts per million in a 1-hour average. In your judgment, what should the standard be?

Dr. BROMBERG. Dr. Lippmann has pointed to the evidence that indicates that the present form of the standard, the one-hour average, may not really be paying adequate attention to the natural patterns of air pollution as they occur in some parts of the country. Also, there is clear evidence that even at less than 0.12 parts per million, with multi-hour exposures, there are significant effects of the type that were originally thought to occur only at levels exceeding 0.12 parts per million.

In reconsidering the ozone standard, it might be possible to retain a one-hour form of the standard, but lower it from the present 0.12 parts per million. Dr. Lippmann and others have sug-

gested that it may be better to put in place a longer term daily standard for ozone which would be lower than 0.12 parts per million.

Senator BAUCUS. Do you agree with Dr. Lippmann on that?

Dr. BROMBERG. Exactly how the experimental observations are going to be translated into action can be debated, but I do not think there is any real question that exposure to ozone at less than 0.12 parts per million in healthy people is capable of causing health effects. Some attention is going to have to be paid to that.

Senator BAUCUS. Thank you both very much for your testimony today.

Our next panel consists of Dr. Thomas Godar, Dr. Philip Landrigan, and Dr. Bailus Walker.

Dr. Walker, please proceed first since I know you have another appointment to make.

STATEMENT OF BAILUS WALKER, PAST-PRESIDENT, AMERICAN PUBLIC HEALTH ASSOCIATION, WASHINGTON, DC

Dr. WALKER. Thank you very much, Mr. Chairman. We have agreed to discuss different aspects of fundamentally the same problem.

I am Bailus Walker, professor of environmental health and toxicology, State University of New York, Albany, and immediate past-president of the American Public Health Association. I was also a member of the National Academy of Sciences' Committee on Epidemiology of Air Pollution. A report of our study has been submitted to the Congress and there are additional copies available at the Academy.

Today I am representing the American Public Health Association, and I am pleased to discuss the health effects of selected air pollutants in non-attainment areas. I think it is fair to say that no reputable member of the scientific community questions the potential risk to human health of air pollution. For all pollutants there is a limit beyond which health and life are threatened. I think this common-sense conclusion is borne out by both accidental exposures of individuals and by certain community exposures.

Let me just comment very briefly on acid precipitation. I will not cover everything that is in my written statement.

Acid precipitation is an old problem whose growth has been insidious. It is now a serious environmental health problem. It is a phenomenon, not a toxic substance per se, but it can increase the risk of human exposure to air pollutants and toxic metals, including sulfur dioxide, nitrogen oxides, and their transformation products, reactive hydrocarbons, as a precursor of ozone.

One measure of the magnitude of the problem is the annual emissions of the major precursors of acid precipitation. Here we are talking about approximately 26 million tons of sulfur dioxide, 21 million tons of nitrogen oxide, and 24 to 28 million tons of hydrocarbons. Over 80 percent of these emissions were emitted from sources in some 31 States.

If we add to that load the estimated 4.8 million pounds of toxins, including 235 million pounds of carcinogens such as benzene and formaldehyde, and some 527 million pounds of such neurotoxins as

toluene and trichlorethylene which are annually released into the air, it is clear that we are confronting a major environmental health problem.

Almost 102 million people in the United States live in counties which exceed one or more of the air quality standards. Several counties in the Far West, the Midwest, and the East, still exceed the ambient standard for sulfur dioxide. Here we know from a very large volume of health effects studies, that excessive levels of sulfur dioxide in the ambient air are associated clearly with acute and chronic respiratory diseases.

Sulfur dioxide is a respiratory irritant that is absorbed in the nose and upper respiratory tract. Public health studies have found an increase in lung cancer mortality in men and an increase in bronchitis in men and women who were exposed to sulfur dioxide. This is long-term exposure.

Sulfur dioxide aggravates existing conditions such as asthma and heart disease. During even brief exposures to sulfur dioxide, asthmatic individuals experience impeded movements of air in and out of the lungs.

At very low concentrations, the individual may experience difficulty in breathing, wheezing most of the day and night. I think exercise certainly potentiates these effects.

Once in the atmosphere, SO_2 is converted to sulfate particles. Two main types are known to be produced: sulfuric acid and ammonium sulfate. Both types are extremely small and may be inhaled into the deep portion of the lungs, as Dr. Bromberg showed in his slides.

Sulfuric acid aerosols have a variety of adverse health effects including spasm of the larynx and of the lungs which cause death, depending upon the concentration and individual sensitivity. Populations at special risk from particulate sulfates are the elderly and adults with pre-existing chronic heart and lung diseases. Children are especially susceptible to increased lower respiratory tract illnesses and to decreased lung function, which Dr. Landrigan will further elaborate on, certainly.

Many analyses of cities with different air pollution levels, comparing death rates among specific populations, have shown a very strong correlation between pollution levels and death rate. The evidence is stronger for the correlation between acute episodic effects and severe air pollution incidence.

The combination of sulfate particles and other associated particles in the atmosphere certainly may cause severe health damage and death. Indeed, this exposure may cause approximately 50,000 premature deaths per year.

Nitrogen oxide, another precursor, refers to a number of compounds: nitrogen oxide, nitrogen dioxide, and such secondary products as nitric acid and nitrate aerosol. Los Angeles County is probably the only county that has not attained air quality standards for nitrogen oxide, a deep-lung irritant capable of producing severe lung damage.

Infants and children exposed to atmospheric conditions in Los Angeles experience a much higher than normal rate of acute respiratory infections. Unfortunately, not enough is known yet about the effects of nitrogen oxides to permit a precise, quantitative esti-

mate of the health-related damages for which NO_x may be responsible.

There are some indirect health effects of acid precipitation which must not be overlooked. Acid precipitation may increase human exposure to several potentially toxic metals by increasing metal concentrations in major pathways, particularly food and water, and in some cases the conversion of metal species to toxic forms.

Acidified waters may dissolve such toxic metals as aluminum, copper, lead, and mercury. It can also release such toxic substances as asbestos from soil and rocks in watersheds, lakes, and drinking water distribution systems.

Of particular concern is lead and mercury. Acidification of drinking water sources significantly increases lead content of our water supplies. Leaded water pipes become particularly hazardous as the pH decreases.

The effects of lead on brain function are increasingly reported. This has prompted the Center for Disease Control to lower the definition of elevated blood lead levels in children.

Mercury can also occur in several species. Of concern here is the methyl-mercury, which has a tendency to bio-accumulate in nature and is dependent to some extent on acidification. The effects of methyl-mercury on the fetus are the best documented examples of metal compounds that affect the developmental process of the central nervous system, including postnatal growth retardation.

In summary, air pollution is one of the greatest risks to public health. It causes, contributes to, and aggravates a long list of diseases and dysfunction: chronic bronchitis, lung cancer, nervous disorders, and heart disease.

The American Public Health Association strongly urges the Congress to strengthen the Clean Air Act with the necessary amendments you will be considering, to allocate the resources needed for the regulatory agencies, both at the Federal and State levels for the enforcement of the Amended Act, and to maintain very careful oversight to ensure that the goals of the legislation are met.

It is very clear that the atmosphere constitutes our most intimate environmental contact. As such, it must promote health and not destroy it.

Thank you.

Senator BAUCUS. We have a vote going on now. The subcommittee will recess for about 10 minutes.

[Recess.]

Senator CHAFEE [assuming chair]. The subcommittee will come back to order.

Dr. Walker, with your time schedule, can you stay through the other panelists and then answer questions?

Dr. WALKER. Yes, I can.

Senator CHAFEE. Fine. Let's move now to Dr. Godar.

STATEMENT OF THOMAS GODAR, AMERICAN LUNG ASSOCIATION, WASHINGTON, DC

Dr. GODAR. Thank you, Mr. Chairman and Members of the Subcommittee.

I am Dr. Thomas Godar, Director of the Pulmonary Disease Section at Saint Francis Hospital and Medical Center in Hartford. I am an associate professor of medicine at both Yale and the University of Connecticut Schools of Medicine. More importantly, I am a practitioner of pulmonary medicine and am here representing the American Lung Association as its President.

The American Lung Association considers that strengthening the Federal Government's Air Pollution Control Program has an extremely high priority for public health. There have been brief comments on the striking advances in information concerning the health effects on humans from experimental exposures to ozone. When the American Lung Association first appeared before this subcommittee some years ago, we presented a state of the art entitled, "Health Effects of Air Pollution," published by the American Lung Association for its medical community, the American Thoracic Society.

An updated version of that report is almost complete. It includes the following key findings that I would simply like to highlight today. With regard to ozone, ozone can cause immediate short-term changes in lung function and increased respiratory symptoms in healthy adults and children who exercise moderately or heavily during periods of elevated ozone levels. There are significant decreases in lung function and symptoms such as cough and pain on deep breathing in individuals exercising for as little as 1 to 2 hours at comparable nonattainment area levels.

Lung function decrements have been found in particularly sensitive exercising individuals exposed to ozone at or below the current standard of 0.12 parts per million. I might add that I consider the one-hour standard to be artificial when you look at human exposure. Significant changes in lung function and increased frequency of respiratory symptoms have been shown to occur at concentrations even below 0.12 parts per million when exposure periods are longer, such as 6½ to 7 hours.

In addition, we have concerns that short-term effects may translate into long-term effects. I am particularly concerned about recurring prolonged exposures in the northeast region and what that might do to the frequency of chronic lung disease in the adults in future years. Our information there is limited, but it is imperative that we take action while we accumulate more information with regard to the result of acute exposures and to what extent they produce chronic disease.

With regard to the health effects of sulfur dioxide, SO_2 at levels as low as 0.25 parts per million for several minutes causes an increase in airway resistance or broncho-constriction in asthmatics who are involved in moderate exercise. Acid aerosols will also cause broncho-constriction in exercising asthmatics near or within the current ambient air standard.

With regard to the health effects of nitrogen dioxide, at low levels of current peak ambient air pollution, there are effects on lung function in asthmatics and there is some correlation between exposure in children and an increased frequency and severity of respiratory infections in both epidemiological studies and confirmed by animal studies.

With regard to populations at risk, the American Lung Association feels that some 62 percent of all Americans, three out of five individuals, breathe air containing pollutants that exceed our ambient air quality standard for one or more pollutants. That is 150 million Americans who are forced to live with air quality that exceeds our health-based standards.

The Clean Air Act requires that standards be set with an adequate margin of safety and also urges that we concern ourselves with the high risk groups. The American Lung Association has been concerned with that for over two decades. We quantify subgroups in a report entitled, "Breath in Danger."

We have estimated the population at risk, using EPA's definition of the population at risk. Let me simply comment on how we define them.

Estimates of the populations at risk in nonattainment areas for carbon monoxide and ozone, for example, include the following. For ozone: persons with pre-existing respiratory disease, emphysema and bronchitis, (commonly known as COPD); elderly individuals 65 years or older who have a relatively deficient immune and defense system; pre-adolescent children 13 years of age and younger, or those who have lung defense mechanisms not fully developed who exercise at high levels and have a higher than normal oxygen requirement and metabolism.

Our estimates for ozone effects are conservative, and for exposure they are quite conservative, because this does not include the large group that we refer to frequently as responders, the particularly sensitive groups, that five to 20 percent of the exercising, apparently healthy normal adults and children who have an increased sensitivity.

For carbon monoxide, our risks group is largely pregnant women, with a risk to the fetus, since carbon monoxide affinity of hemoglobin is considerably greater than that for oxygen, and also persons with pre-existing coronary heart disease who may not have a lung deficiency, but in whom carbon monoxide basically robs the red blood cells of oxygen-carrying capacity in a patient with a critically reduced circulation to an exercising muscle.

Our analysis for counties classified as not in attainment for ozone and carbon monoxide estimates the magnitude of the population at risk as follows: 354 counties that are either classified as not in attainment for ozone or so proposed. That is 123 million persons: 25 million pre-adolescent children, 15-plus million elderly, just under 5 million asthmatics including children, and 6 million with Chronic Obstructive Lung Disease. That is our at-risk population.

There are 214 counties that are either classified as not in attainment for carbon monoxide or proposed for such classification. That includes 89 million people within that group. The at-risk group would be 1.8 million with coronary heart disease and 1.4 million pregnant women.

The recent EPA air quality monitoring data for 1988 adds further concern to the American Lung Association because of the quality of our air. It is actually not improving in some areas. It is deteriorating.

We have added 28 new areas with ozone levels above the health standard. The subgroups at risk are 2.9 million pre-adolescent chil-

dren, 1.7 million elderly, as well as 900,000 asthmatics and over 900,000 with Chronic Obstructive Lung Disease. We have summarized some of this in our report entitled, "Breath in Danger."

To make some general comments about the health costs——

Senator BAUCUS. I am going to have to ask you to summarize your statement if you could, Doctor.

Dr. GODAR. Yes, sir.

We have prepared a document entitled "The Health Costs of Air Pollution," in August 1985, and in our prepared statement are some numbers indicating the billions of dollars that are being spent in managing medical care secondary to air pollution exposure as well as in terms of morbidity and mortality for both children and adults. I would also like to draw your attention to the fact that in the submitted testimony, we indicate our concern that more attention must be given to the control of nitrogen oxides as a major mechanism for clean air, not only to affect particulates, acid aerosols, but also a major precursor for ozone.

In conclusion, the American Lung Association believes that to provide a strong Federal, State, and local decisionmaking process and direction, we require a strong reauthorized Clean Air Act. This must include new emission standards for autos and trucks using maximum current technology available, new deadlines for attainment areas to achieve the health-based standards, requirements that State implementation plans for ozone include nitrogen oxide controls, requirement for 90 percent controls on motor vehicles and power plants, and finally, programs to attack ozone nonattainment on a regional basis, particularly in the severely polluted northeast region of this country.

The American Lung Association stands ready to help you in any way we can, to provide data, support, and to offer our scientific and medical expertise. Thank you for the opportunity to speak to the committee today.

Senator BAUCUS. Thank you, Doctor.

Senator Chafee, do you have any questions?

Senator CHAFEE. Yes, Mr. Chairman. Unfortunately, I must go to another committee hearing.

I assume that we have a copy of that "Health Costs of Air Pollution" in the committee. If not, could we get one from you, Dr. Godar?

Dr. GODAR. Yes, sir.

Senator CHAFEE. Dr. Walker, you come up with some pretty powerful allegations here. On page three of your testimony you say, "In summary, air pollution is one of the greatest risks . . . as many as 50,000 premature deaths may be caused by single air pollutants or a combination of pollutants." Is that 50,000 per year?

Dr. WALKER. That is an annual figure of 50,000 per year.

Senator CHAFEE. Per year? All we have tossed around here was a figure of 2,000, discussed here earlier today.

What is that figure based upon?

Dr. WALKER. Several sources, sir. The Office of Technology Assessment did an analysis, and some of that data comes from their announcement.

Senator CHAFEE. There is another statistic you used. On page two, you say the same thing, but there you say 50,000 premature deaths in Canada and the United States.

Dr. WALKER. You are correct, sir. That does include both Canada and the U.S.

Senator CHAFEE. These are really startling figures. I wonder if you could write to me and give us the source of those statistics? I would appreciate it.

Dr. WALKER. Yes, I will do that.

Senator CHAFEE. That is very potent material.

Thank you, Mr. Chairman.

[The information requested follows:]

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May 8, 1989

Honorable John H. Chafee
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Dear Senator Chafee:

On April 18, 1989 when I presented testimony on behalf of the American Public Health Association before the Senate Subcommittee on Environmental Protection, you expressed interest in our estimate of health risk -- 50,000 premature deaths in the United States and Canada -- posed by acid precipitation precursors.

For these estimates, we drew heavily upon data assembled by the Office of Technology Assessment (OTA).

First, the process for acid deposition begins with emissions of sulfur dioxide (SO_2), nitrogen oxides (NO_x) and hydrocarbons. Air pollution episodes characterized by particulates and high levels of SO_2 have resulted in increased deaths in people with pre-existing heart and lung diseases. Here, it should be emphasized that air pollution can be related to disease in three basic ways: as a cause; as a contributing factor; or as an aggravating factor. Another related consideration is that human populations are exposed to a large number of air pollutants, many of which may interact to produce a health effect greater than the sum of the effects of each pollutant.

There is substantial evidence of injury from some aspects of the sulfate-(once in the atmosphere SO_2 is converted to sulfate particles)-particulate mix in air pollution. At high exposure levels, sulfur-particulates in air pollution can aggravate asthma, chronic bronchitis, and heart disease. There is also evidence that sulfur-particulate air pollution causes acute respiratory disease in children.

Moreover, many studies of communities with different air pollution levels, comparing death rates among specific population groups or cohorts, have shown strong correlations between pollution levels and deaths. Evidence is strongest for correlation between acute episodic effects and severe air pollution incidence; evidence of long-term effects at comparatively low levels of pollution is more limited. Indeed, the respiratory health of the public is not improving as is evident from death rate of diseases of the respiratory system excluding cancer of the lungs, according to two decades of data from the National Institutes of Health.

A model to estimate the number of excess deaths due to sulfurous air pollution has been developed by scientists at the Brookhaven National Laboratory. They estimated that 50,774 excess deaths (with a 90% range of 0 to 159,950) in the United States and Canada could be attributed to acid rain. In Canada, the number of excess deaths was 4,076 (with a 90% range of 0 to 12,125). The model used to develop these estimates is a complex function of both local and long-range air pollution, regional population patterns, and many other demographics.

It is in this setting that OTA developed mortality estimates. OTA used SO_2 emissions data, population densities and made several assumptions to develop estimates for excess deaths due to sulfate-particulate air pollution (sulfate concentration is used as an index of sulfur-particulate air pollution) for three scenarios: (1) 1980 population levels using 1978 emissions data; (2) population in 2000 assuming that total emission levels are held constant at 1978 levels; and (3) population at year 2000 assuming that sulfate-particulate levels are reduced by 30 percent from 1978 levels. A summary of the results is below:

Scenario	Population (millions)	Excess deaths total(10^3 deaths)	Range	Rate(deaths/10^5 population)
1.1980 population, 1978 emissions, U.S. and Canada	249	51	0-150	20.4
2.2000 population, emissions unchanged, U.S. and Canada	291	57	0-170	19.8
3.2000 population, sulfate-particulate mix 30% below 1978 levels, U.S. and Canada	291	40	0-120	13.8

Source: OTA

Total pollutant emissions in the United States and Canada have been estimated to result in about 50,000 premature deaths under the first scenario.

In the United States, approximately 2 million persons still reside in counties where the SO_2 levels in 1987 exceeded the primary National Air Ambient Air Quality Standards. In terms of air quality emission trend between 1983-87, SO_x emissions decreased by only 1 percent. Revisions to the sulfur dioxide standard requires further stringencies on current emission limits on existing sources, and major changes in air quality implementation plans in some states.

I trust this information is a response to your request.

Respectfully,

Bailus Walker, Jr., Ph.D., MPH
Professor of Environmental
Health and Toxicology

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Senator BAUCUS. Thank you, Senator.

We are joined here now by the Majority Leader, Senator Mitchell.

Senator Mitchell, any questions or comments?

Senator MITCHELL. Thank you very much, Senator Baucus. I commend you for holding this hearing because I think it raises very serious questions.

In 1987, both Dr. Landrigan and Dr. Godar testified at a similar hearing. Dr. Walker's predecessor was at that hearing, or another representative of the same association.

At that hearing, the transcript of which I have before me, all of the four witnesses at that time testified that acid rain should be controlled solely on the basis of health effects. That is, the adverse health effects of acid rain were sufficient to justify acid rain controls, notwithstanding any additional damage that might occur to the natural environment.

I would like to ask Dr. Godar and Dr. Landrigan if they still agree with that conclusion, and Dr. Walker, if he now agrees with that conclusion?

Dr. Godar.

Dr. GODAR. Yes. I received some alarming comments from our Department of Environmental Protection. We have considered ozone levels in Connecticut to be bad indeed and the entire State is not in compliance as a State. We are the recipients of the precursors from our neighbors and also generate plenty of our own. We have always considered the photo-chemical smog as largely ozone.

We are now informed that the DEP feels strongly that there is a significant component of acid aerosol. I find that very distressing since you put SO_2 in higher forms together with ozone, which is an oxidant, a very reactive chemical, and there can not be anything but synergistic effects, at least from what we know.

I am very concerned that what we have treated as pure ozone in an area where ozone is a predominant pollutant may in fact be substantially acid aerosol. Some of this may well be nitrogen oxide-derived, as well as sulfur oxide. I am concerned that it is part of what is reacting in the upper/lower respiratory tract.

Senator MITCHELL. Dr. Landrigan.

Dr. LANDRIGAN. Yes, I still stand by that position, Senator. The effects of acid air pollutants including ozone on the health of children are severe.

If cost estimates are needed to buttress the health position, the American Lung Association has done econometric studies that show that the costs of lung disease caused by pollution run into the billions per year. That is only the financial costs. It doesn't even speak to the human costs, the pain and suffering. I think that the position is well justified on health grounds alone.

Senator MITCHELL. Dr. Walker.

Dr. WALKER. Senator Mitchell, I certainly support that based on what we know about the precursors of acid deposition. If we are to put emphasis on prevention, I think we should control it effectively.

Senator MITCHELL. Dr. Walker, in your further response to Senator Chafee's questions, I would appreciate it if you would include an answer to the following additional question.

If in fact there are 50,000 premature deaths per year in the United States and Canada, if we fail to act, to deal with air pollution, do you anticipate that the number will increase, and if so, to what extent, if that can be predicted with reasonable reliability?

Dr. WALKER. Yes, sir.

[The information requested follows:]

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June 7, 1989

The Honorable George J. Mitchell
United States Senate
SR-176 Russell Senate Office Building
Washington, D.C. 20510-1902

Dear Senator Mitchell:

On April 18, 1989 when I presented testimony on behalf of the American Public Health Association before the Senate Subcommittee on Environmental Protection, you expressed interest in future trends of air-pollution-related disease, dysfunction and premature deaths. "Approximately how many premature deaths can we expect if we do not take action" was your question. You requested a written response which is within the following pages.

In my testimony I cited a current estimate of 50,000 premature deaths per year in the United States and Canada as the health risk posed by airborne sulfates alone. This estimate was derived from data developed by the Office of Technology Assessment (OTA). OTA drew heavily on a combination of previous estimates and it made several assumptions about levels of pollution, population growth and shifts. If appropriate Congressional action, or voluntary action by polluters, is not taken to reduce human exposure to a broad range of toxic air pollutants, then we can expect substantial increases in the incidence of air-pollution provoked disease, dysfunction and premature deaths.

A two- to three-fold (at least) increase, or <u>100,000 to 150,000</u> premature deaths per year would not be an unrealistic estimate. It is in fact a most conservative estimate. To be sure, the convergence of at least seven factors is setting the stage for this increase.

<u>First</u>, among the working-age population, respiratory disease, coronary heart disease and stroke already represent a

significant burden in terms of morbidity and mortality. Many members of this group suffer not only acute illness requiring hospitalization and a physician's care but chronic and disabling conditions that may require long term care. Here, it should be pointed out that an increasing number of persons in this age group are engaged in some type of outdoor exercise (jogging, bicycling, walking for example). As physical exertion draws more air into the deep lungs, it draws more pollutants. And not all persons heed the warning of health authorities to stay indoors when air pollution levels are "high."

Second, there is an estimated 30 million plus Americans without adequate health care to promote early detection and treatment (prevention of progression) of degenerative (or potentially degenerative) diseases which are further exacerbated by exposure to air pollution.

Third, the increasing number of newborns with defective immune systems and multiple chemical burdens, due in part to parental exposure, is adding to the group of persons who are at highest risk of air-pollution-related disorders.

Fourth, the above demographics are underscored by data which show that more than half of the nation's population live in communities where at least one of the six pervasive pollutants -- ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, particulates and lead -- exceed ambient air quality standards.

Fifth, large segments of the population are exposed to increasing concentrations of acid aerosols for which there are no EPA standards. These tiny particles transport the most toxic substances into the deepest regions of the respiratory tract. In the process, they can cause irritation of the air passages which results in constriction of the airways. Edema (accumulation of fluids) may occur and secondary infection frequently compounds the damage, and may ultimately result in death.

Sixth, EPA-established standards are obviously not adequate to protect the general population with a "respectable" margin of safety. Indeed, the existing standards are not protective of the more than 8 million asthmatics and millions of other persons who are sensitive to a broad range of air pollutants -- both indoor and ambient.

Seventh, less than 5 percent of the hundreds of hazardous pollutants Congress targeted for control at their source are being regulated. These pollutants increase the risk of cancer, birth defects, genetic and other degenerative or

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irreversible illnesses with a high fatality rate.

The estimate of 100,000 to 150,000 premature deaths also takes into consideration the experiential reality that air pollution is related to disease, dysfunction and premature death in three basic ways: as a cause, as a contributing factor, or as an aggravating factor.

There are other data that lends weight to our estimate. The respiratory tract presents a large area in direct contact with environmental pollutants and thus it is the human system at highest risk of injury due to acid precipitation and other airborne pollutants. As a result, respiratory disease constitutes a major public health problem as measured by the number of persons affected, the number of days of productive activity and years of productive life lost (premature deaths) and the direct cost of caring for persons suffering from them.

According to the National Institutes of Health, lung diseases are a leading cause of death and disability in the United States. Nearly 240,000 deaths each year are directly attributable to lung disease, and these diseases are contributing to many additional deaths. Thus, lung disease may cause <u>one out of every eight deaths</u> and play a significant role in <u>one out of every four</u>.

Almost 18 million Americans have chronic bronchitis, emphysema or asthma, and more than 100 cases of influenza, pneumonia and acute bronchitis occur annually. Respiratory disease accounts for approximately 2.5 million hospital discharges, 21 million days of hospital care and 25 million physician visits. Lung disease accounts for more workdays lost (more than 31 million annually) than any other category of illness. To this impact must be added the devastating psychological and personal economic effect of a chronic incurable respiratory disease. Progressive respiratory impairment results in a decreasing ability to carry on usual activities of daily living. The impairment may eventually lead to severe limitation of function and loss of earning capacity and dependence of public assistance. Worry and anxiety may produce intense stress for both the patient and the family and could well cause premature death of the patient.

Unfortunately, the data we now collect cannot precisely alert us to the longer-range trends for individual disease nor to the interactive or accumulative effects of all air pollution-related respiratory disorders. One barrier to more precise data collection is the fact that respiratory disease typically presents a blurred picture to the diagnostician. Treating the symptoms pragmatically perhaps offers relief but

leaves an enormous amount of disease imprecisely diagnosed or unreported. As a result, we know very little about the overall cost of respiratory disease. What is clear, however, is that much of the respiratory disease problem is preventable, particularly that which is related to indoor and ambient air quality. Thus, substantial reductions in present air pollution levels would prevent a significant number of premature death, and also reduce the cost of health care.

Senator Mitchell, I trust this is an adequate response to your inquiry.

Sincerely,

Bailus Walker, Jr., Ph.D., MPH
Professor of Environmental
Health and Toxicology

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Senator MITCHELL. Mr. Chairman, I think it is very significant that the American people, through the press that are here, grasp the significance of what has been presented here today.

We have been told by persons expert in the field, whose prior testimony has never been rebutted or even disputed—it is important to note that they testified to similar effect 2 years ago, and there has been no intervening dispute or rebuttal of their testimony to my knowledge—but now we have been told that associated with air pollution in the United States and Canada are 50,000 premature deaths each year, 25 million children at risk, a 25-percent increase in asthma in children, 15 million elderly persons at risk, and health care costs of up to $40 billion each year due to air pollution.

It is difficult for me to reach any conclusion other than that this is a public health emergency, an emergency of national scope and effect, requiring prompt and vigorous national action to deal with it. I think if every American parent comprehended the threat to his or her child from air pollution, they would overwhelm Congress with the demand for action.

The only missing link is our inability to establish cause and effect between a specific source of pollution and a specific pollutant and the health of a specific child. We know in the aggregate what is occurring. A large number of sources emitting a large quantity of pollution are causing a large amount of damage to a large number of Americans. It seems to me that of all the testimony that we have received here on this subject, what we have heard of the effects on the health of the American people is the most important, the most compelling, and serves as the basis for action in this Congress.

Mr. Chairman, I conclude by thanking you and Senator Lieberman for permitting me to go out of turn here, and I commend you again for your leadership in this area. I do reaffirm my intention to have action in this Congress on this matter. I think that if every American citizen heard and comprehended what has been said here today, they would demand action from the Congress.

Thank you very much, Mr. Chairman.

Senator BAUCUS. Thank you, Senator.

Dr. Landrigan, we would like to hear your statement now.

STATEMENT OF PHILIP J. LANDRIGAN, AMERICAN ACADEMY OF PEDIATRICS, WASHINGTON, DC

Dr. LANDRIGAN. Thank you, Mr. Chairman and Members of the Committee.

I am a pediatrician and also professor of pediatrics and environmental medicine at Mount Sinai, and I am here this morning representing the American Academy of Pediatrics. The Academy of Pediatrics is concerned about the effects of air pollution on children. We estimate that more the 28 million children in the United States reside in communities where clean air standards have not been achieved for the criteria of pollutants, including ozone, carbon monoxide, and acid air pollutants.

To put things in perspective, it is true that ambient concentrations of many air pollutants, such as sulfur dioxide and particulates have decreased sharply over the past two decades since the

original passage of the Clean Air Act in 1970. However, against that downward trend, unfortunately in the past few years, there have been countervailing upward trends for concentrations of two air pollutants, oxides of nitrogen and ozone. It is about those two effects that I would like to speak particularly this morning, focusing on their effects in children.

The most recent EPA data for the year 1988, which was released about a month ago, indicates that in 1988 there were 26 new urban and two new rural areas in the United States where the national ambient air quality was exceeded for the first time. Another point that came out in those data was that 1988 was the third year in the past decade in which national ozone records have broken all records.

Ozone is a very highly reactive gas. When it is inhaled, it functions as a very potent respiratory irritant. You saw Dr. Bromberg's slides earlier this morning, and they illustrate the effect of ozone on the lung.

It causes inflammation, irritability of the airways, coughing, chest pain, tightness of the chest, and can provoke asthmatic attacks. It is worth noting in this context that the rate of hospital admissions for asthma over the past decade have increased approximately 50 percent in this country. Also, the number of deaths from asthma have risen over this same period of time.

One of the things that has become clear in the studies of the health effects of ozone and other acid air pollutants is the fact that not all people are equally susceptible. I believe that all people probably react at some level and all people react if doses are high enough, but within the population there is certainly variation.

One of the most susceptible groups are the children. The elderly are another susceptible group.

The reason children are vulnerable is a direct function of their anatomy. Children have narrow airways. There airways are much narrower than those of adults. Thus, minor inflammation that causes some swelling of the lining of an airway in a child and some narrowing of the passage has a much more serious effect in a child than it would in an adult.

A child simply does not have the anatomic or functional reserve that an adult has. An amount of inflammation, a narrowing of the airways, that would be trivial in an adult can produce profound respiratory distress in a child and provoke an asthmatic attack.

Another reason why children are more vulnerable than adults to air pollutants is that they have respiratory infections more frequently. The interaction of these infections with pollutants such as ozone intensifies the damage that the pollutants cause to the lungs.

A lot of work has been done to try to assess the level of exposure to various pollutants such as ozone, which are associated with damage. This sort of work is necessary, of course, to provide a numerical basis for any effort to change the standard.

One of the things that has been seen in research conducted by EPA is that exposures to volunteers in chambers—and these were adult volunteers, not children—produce respiratory effects at concentrations as low as 0.08 parts per million. I must point out that this is a level that is only two-thirds the present standard of 0.12. If those findings are corroborated, that would suggest that the stand-

ard ought to come down at least to a level of 0.08 and perhaps lower if a built in margin of safety is desired.

The Harvard-Six-City study about which you have heard much and will hear more, as well as the studies conducted by the New York University-Rutgers group among children in summer camps in New Jersey, New York, and Pennsylvania, have also shown that ambient concentrations of ozone at or even below the current standard are associated with pulmonary deficits in children. One of the issues that always comes up in discussing those deficits is whether they truly represent something of medical importance or if they are just some sort of metabolic adjustment that the lungs make to the presence of some pollution in the air.

I think that the best data to address that troubling issue are the data which were mentioned earlier this morning. These come from the studies done in Los Angeles, looking at the chronic effects on pulmonary function of residents in a part of Los Angeles that had heavy air pollution as compared with pulmonary function of those people who reside in a part of the city with light air pollution. Those studies were done in non-smokers, so the issue of smoking interfering with the results does not apply.

The studies show very clearly that people who reside in the polluted parts of Los Angeles have rates of loss of pulmonary function over the period of the five to ten year span which are substantially greater than the rates of loss of function in people who live in less polluted parts of the city. What that means in plain language over the long haul is that people, even non-smokers, who reside in heavily polluted areas are going to show a higher rate of pulmonary deterioration than people who live in clean parts of the country.

Therefore, Mr. Chairman, on behalf of the American Academy of Pediatrics, I would like to urge you and the Members of both Houses of the Congress to take a hard look at the present strategies for ensuring clean air in this country. It is our opinion that the present strategies, although they have had some limited successes, in the aggregate, are not working. The increase in ozone and oxides of nitrogen which have been seen in recent years are having a definite and permanent negative effect on the health of our Nation's children.

Thank you.

Senator BAUCUS. Thank you very much, Dr. Landrigan.

Dr. Godar, the American Lung Association recently issued a statement calling on a report that showed the adverse health effects of nitrogen oxides. I wonder if you could expand upon that a bit and focus on the salient points of that report—that is, the degree to which, in the opinion of the American Lung Association, nitrogen oxides do present a health hazard?

Dr. GODAR. First of all, the nitrogen oxides are unique in that they are clearly precursors for ozone, perhaps the most important precursor along with hydrocarbons. It is itself a criteria pollutant.

It has inflammatory effects on the airways and has been correlated in areas of high levels with increased respiratory symptoms and perhaps increased incidence of chronic respiratory disease in children in urban centers. It is part of the mix resulting in acid aerosol along with sulfur oxides. It is also a particulate which permeates all of the air pollution problems we have.

Yet, it has received the least attention. Sometimes what receives the most attention is the thing easiest to measure or most prevalent, for example in many cases, ozone. Or it may be something that has an acute effect which draws attention.

Nitrogen oxide has fallen by the wayside in terms of our focus. We believe it is extremely important to get back to attainment for NO_2, and looking at that in ways to control NO_2 as a major mechanism for control of both acid aerosols and for control of ozone.

Senator BAUCUS. So you would rank it on par with efforts to control various other criteria pollutants?

Dr. GODAR. Yes.

Senator BAUCUS. The basic bottom-line question is the degree to which present standards and present law provide an adequate margin of safety for public health. I would like each of you to address the degree to which you would change current standards so that we do have, in your judgment, an adequate margin of public health protection.

Why don't you begin, Dr. Godar?

Dr. GODAR. Let me take a conservative approach, given the fact that a good part of the Lung Association's membership represents the scientific community, the Thoracic Society.

Scientific investigators tend to be extremely scrupulous in terms of their data. They will not accept concepts until they are not only demonstrated, but also redemonstrated several times over, to their satisfaction. They take a very conservative position and, I think, justifiably so, with regard to air pollution health effects.

Those of us who are clinicians are inclined to see this anecdotally every day and are somewhat less conservative. I will take a middle position and say that I am concerned that we have not met ambient air quality standards, rather than changing the standard. I was upset that ozone was changed from 0.08 to 0.12 because I felt that there was already evidence that certain sensitive individuals reacted under certain conditions at levels of 0.08, as Dr. Landrigan pointed out.

Senator BAUCUS. You were upset with the change from 0.08 to 0.12?

Dr. GODAR. I was concerned with the improvement in the statistics by changing the standard from 0.08 to 0.12. That was particularly important in Connecticut where ozone is our major pollutant.

I was concerned with that, but would not wish the debate to focus on changing the ozone standard necessarily. I would like to focus on the fact that we have failed completely to attain the standards that we currently have, based on the available scientific information.

We have enough information. We already have good evidence that we should be making every effort to attain the standards we have set. I would like to see the emphasis in terms of a renewed forceful approach to achieving our current standards, rather than debate over whether the ozone standard might be modified up or down.

Senator BAUCUS. Is attainment of current standards sufficient in your judgment?

Dr. GODAR. No.

Senator BAUCUS. So you believe that first, we should reach the current standards?

Dr. GODAR. Absolutely.

Senator BAUCUS. Second, if we were to do so, to what degree would you raise the standards for ozone, for example?

Dr. GODAR. I would be interested in ongoing research to demonstrate more firmly that there is in fact a large sensitive population that requires 0.08 or less to be safe, to provide what the Clean Air Act should provide, which is a reasonable margin of safety for the most sensitive group. We are not doing that currently.

Senator BAUCUS. Dr. Landrigan.

Dr. LANDRIGAN. I would argue that the standard ought to be reduced to the level of 0.08. I would also argue that there has to be careful attention paid to the nitty gritty issues of the standard. It is not enough to simply set a number. The regulators have to think carefully about the duration of averaging time. Should it be set at 1 hour, 6 hours, or 8 hours?

Senator BAUCUS. Should we have a longer standard on that?

Dr. LANDRIGAN. I defer to Dr. Lippmann on that. He mentioned earlier this morning his opinion that it should be eight hours, and I would tend to concur with that.

The third thing is enforcement. All the numbers in the world mean nothing if there is not vigorous hard-nosed enforcement.

Senator BAUCUS. Do you have any views on other criteria pollutants other than ozone? Should the standards be tighter?

Dr. LANDRIGAN. I will defer on that. I have thought principally about ozone.

Senator BAUCUS. Dr. Walker, should the standards be increased?

Mr. WALKER. I would agree with Dr. Godar in that I think we first ought to attain the standards that are now in place. I also think that we ought to recognize that the standard-setting process is never complete. There must be ongoing evaluation of these standards in terms of new data that may be coming out from the research laboratories.

I would agree in terms of the ozone standard. I have no opinion on some of the others. I do think that attainment of current standards is a very critical issue.

Senator BAUCUS. Thank you.

Senator Lieberman.

OPENING STATEMENT OF HON. JOSEPH I. LIEBERMAN, U.S. SENATOR FROM THE STATE OF CONNECTICUT

Senator LIEBERMAN. Thank you, Mr. Chairman. I regret that I could not be here at the beginning of this hearing. I do have an opening statement and would ask unanimous consent to have it included in the record.

[Senator Lieberman's statement follows:]

OPENING STATEMENT OF HON. JOSEPH I. LIEBERMAN, U.S. SENATOR FROM THE STATE OF CONNECTICUT

Thank you, Mr. Chairman, for holding these hearings.

The subject we address today is the invisible threat of air pollution, and how it affects our health, and particularly those most at risk in our society: our children, our elderly, people with respiratory and heart disease and pregnant women.

The statistics on ozone levels from the summer of 1988 are staggering. The 1988 peak ozone levels were the highest of this decade. Some 150 million Americans (62 percent of all Americans) now live in areas that last year exceeded EPA's maximum safe level of ozone pollution.

Yet despite these statistics there are some who claim that even existing clean air standards are too stringent. Just a few weeks ago, a front page article in the New York Times suggested that the costs of cleaning up the air may not be worth the effort.

Happily, the American public does not agree.

The main reason environmental protection has taken center stage on the agenda of Americans is that the people understand that the pollution of the environment can hurt them, and can hurt their families.

Ozone pollution causes decreases in lung functioning, respiratory infection, coughing and wheezing. And the evidence is mounting that ozone exposure causes permanent lung damage. Its effects on the lungs are similar to that of the effects of mustard gas, the Workd War I poison.

Researchers at Harvard University have concluded that acid rain and its precursors contribute to approximately five percent of the premature deaths in the United States—1 death out of every 20.

Exposure to even modest levels of carbon monoxide can pause permanent brain damages in fetuses.

The New York Times article I mentioned earlier talks about the cost of cleaning up the air. There is a cost, but there is also a high cost associated with dirty air. The testimony of Dr. Thomas Godar, a resident of my home state of Connecticut and the President of the American Lung Association, points to estimates of enormous economic benefits (in the tens of billions of dollars) to society resulting from control of air pollution. One study cited by the Lung Association showed a 60 percent reduction in sulfur dioxide alone would save up to $40 billion in health care and other costs of illness and death related to these pollutants. Another study showed that programs to reduce average particulate and sulfate air pollution in six urban California regions would save 850 people each year from premature deaths resulting from air pollution. Mortality health cost benefits from those programs would be $400 million per year. A third study shows that reducing the average nationwide concentration of sulfur dioxide by one microgram per cubic meter would result in a reduction in morbidity health costs of $20 million per year.

What is at stake is not simply a desire that those of us who are fortunate enough to be healthy continue to be healthy. The Clean Air Act must protect society's most vulnerable and sensitive groups. Dr. Godbar's association recently published a report showing that approximately 60 percent of these most vulnerable people live in areas where air pollution exceeds health standards.

This is a problem that cries out for a federal response, and that response must take the form of a strong Clean Air Act which can mandate reductions in the emissions that threaten all our lives. It will mean changes in the way we live and in the way we do business, but the American people are prepared to make those changes if we provide the leadership. Nothing less than the health of our citizens is at stake.

Senator LIEBERMAN. I also want to welcome the members of this panel, and if I may be somewhat parochial, particularly welcome Dr. Godar, who practices at Saint Francis Hospital in Hartford, Connecticut. We are very proud that he has taken this national leadership role in the fight for cleaner air.

Mr. Chairman, I thought that the questions and the statement from our distinguished Majority Leader were very powerful in summarizing some of the results of the testimony that has been offered by these three experts, both today and in past years, about the health benefits of controlling air pollution. I think we are coming to understand that environmental protection these days equals health protection.

Environmental protection is not simply a matter of protecting our natural resources, as important as that is. It is also a question of protecting our human resources.

I appreciate the contribution that the three of you and others have made to that position, that clarity.

Dr. Godar, I am very interested in the studies that have been cited by the American Lung Association concerning the health benefits of controlling air pollution because they make the point that, not only is there a cost to cleaning the air, but also to not cleaning up the air. There was an article recently in the New York Times which struck many of us and raised the question about whether the cost of cleaning up air pollution was worth it.

I would like to ask if you could briefly review some of the work that the Lung Association is doing now to try to quantify the health benefits associated with the control of air pollution?

Dr. GODAR. I am not an economist and, therefore, least familiar with those kinds of figures. As part of the testimony that I really did not concentrate on, however, there are some numbers which represent figures obtained from "The Health Costs of Air Pollution," which was published by the American Lung Association in 1985.

Some of those numbers will change as a result of inflation and better data, but it did review the current evidence in terms of economic costs related to health problems in this country. The total health cost from exposure to criteria pollutants was considered to fall in a broad range of tens of billions of dollars.

I recently had reason to review the data from the National Center for Health Statistics on Chronic Obstructive Pulmonary Disease alone. The total cost increased 10 percent from 1986 to 1987 and reached $44.6 billion. This was broken up almost evenly into medical costs: hospitalization and medication, and then morbidity costs: lost time from work, and finally mortality costs: lost days and years of working lifetime.

That $44.6 billion is a rather large figure. And that is only one form of Chronic Obstructive Lung Disease that is very significantly affected by exposure to air pollutants, albeit, it may not have been produced primarily by air pollutants. In many cases, it was produced primarily by cigarette smoking.

That is a large population. My own opinion is that it is underdiagnosed and the population base is probably even larger.

They are very unstable and highly sensitive to low levels of pollutants. I think the morbidity and medical care costs may well be in excess of the $44.6 billion that has been estimated.

In the testimony submitted, we do have some numbers in terms of billions of dollars of morbidity and mortality that could be saved by changing specific pollutants. I will not go into those details now, but in examples cited even within this country, such as in Florida, for example, there was a 50 percent savings by simply controlling effluents from sources in four or five regions in Florida. The cost was well below the savings anticipated from health care costs alone.

Senator LIEBERMAN. Needless to say, those are startling numbers—$44.6 billion is far beyond any of the estimates that I have seen of the costs of the Clean Air Act. I assume that they do not include what might be called pain and suffering. They are more direct.

Dr. GODAR. Those figures allude specifically to Chronic Obstructive Pulmonary Disease and would exclude asthma and other

causes. They do not relate directly to air pollution alone, but in fact, our estimates do not include pain and suffering at any time.

Senator LIEBERMAN. Thank you.

Dr. Landrigan, you have testified and your research indicates that prolonged exposure of children to ozone may lead to premature aging of the lungs. I wonder if you could explain to us a little more precisely what actually happens during premature aging of the lungs and how it will affect the ability of our kids to lead normal lives?

Dr. LANDRIGAN. As children grow up to the time when they stop growing at the age of about 18 or 20, their lungs are growing each year along with the rest of their body. There is a normal rate of increase of lung function through childhood and through adolescence.

One consequence of chronic exposure to air poliution in childhood is that this normal rate of increase is truncated so that children do not reach their full endowment. They end up with smaller lungs at the age of 20 years than they ought to have.

Then beyond the age of 20 years, each of us as we age loses some small amount of pulmonary function each year. This is normal and we all do it. However, it has been well described that in people who are smokers or in people who are exposed occupationally to air pollutants, the rate of loss of lung function is greater than the normal, two, three times, even many times greater than the norm.

The recent data from Los Angeles to which I alluded would suggest that the same thing can happen in people, even nonsmokers, who are exposed to air pollutants, ozone and particulates and the whole mix of urban air pollutants year in and year out. They lose pulmonary function at an accelerated rate. That is what I am referring to when I talk about accelerated aging of the lung.

The long-term consequence of that is that if the pulmonary reserve is reduced to a point where 50 or 60 percent of the lung capacity is lost, then the person who was previously vigorous reaches a point where he can no longer walk across the room, and climbs stairs only with difficulty.

Senator LIEBERMAN. It seems to me, Dr. Landrigan, that one of the natural conclusions from your research is that parents should do something that is counter-intuitive, which is to say that there are nice summer days on which it would be better for us to keep our kids inside. What do you advise parents about how to make that determination and when to decide that their kids running around outside is not so good for their health?

Dr. LANDRIGAN. It certainly is counter-intuitive. I think all of us grew up with the notion that playing outside in the summer was probably as close to heaven as we achieve in this earth, particularly in New England. These data which show that the ozone concentrations reach their peak on the sunny days of summer are very distressing.

I have not yet reached the point where I tell people to keep their kids inside, with the exception of the very worst days when the weather bureau puts out their pollution advisories. I have urged my own kids to not do very vigorous things during the hot, stagnant hours of the day—not to do long-distance running, rowing,

those types of things. They can defer those things until the evening hours when the levels have come down somewhat.

Senator LIEBERMAN. Thank you, Mr. Chairman.

Senator BAUCUS. Thank you, Senator.

I also think the best advice we can give those mothers is to not only be concerned about children playing inside or outside in the short term, but more importantly, those mothers should talk to their House Members and their Senators to make sure we get the air cleaned up so the ozone levels will be reduced. That is where we should be placing our effort.

Dr. LANDRIGAN. That's primary prevention, not after the fact.

Senator BAUCUS. I do not want to put words in the mouths of the panelists, but I think it is important due to your expertise and the degree to which you have studied this problem, to know in your judgment the degree to which we have a public health crisis facing America due to air pollution.

Was that a fair statement to make, that we have a public health crisis due to air pollution, or would you phrase it differently? I would like you each to answer quickly, please.

Dr. Godar.

Dr. GODAR. I think that is true. I have before me something not submitted, a document which was printed in February, 1989, showing the number of ozone violations in the Northeast States in 1987 and 1988, which includes New England, New York, and New Jersey.

Most States went up significantly between 1987 and 1988 and Massachusetts tripled its ozone violation days up to 28 for that particular summer of 1988. Maine and New Hampshire tripled their violation dates in 1 year. Vermont appears on the graph for the first time in 1988. I consider this graph frightening, hair-raising, and truly a crisis.

Senator BAUCUS. Thank you.

Dr. Landrigan.

Dr. LANDRIGAN. I agree.

Senator BAUCUS. Dr. Walker.

Dr. WALKER. I agree. If we were only talking about one or two pollutants, maybe not, but we are talking about a whole range of air pollutants.

Senator BAUCUS. Thank you all very much. We appreciate your testimony.

Our final panel consists of Dr. Homer Boushey, University of California at San Francisco; Mr. Michael Schonbrun, the National Jewish Center for Immunology and Respiratory Medicine at Denver, Colorado; Dr. Robert Phalen, University of California at Irvine, California; Dr. Christopher Green, General Motors Research Lab, and Dr. Regis McFadden, University Hospitals of Cleveland at the request of the American Mining Congress and the National Coal Association.

Dr. Green, why don't you go first?

STATEMENT OF CHRISTOPHER GREEN, GENERAL MOTORS RESEARCH LABORATORY, WARREN, MI, ACCOMPANIED BY ELAINE S. WRIGHT, PULMONARY TOXICITY RESEARCH PROGRAM MANAGER

Dr. GREEN. Thank you, Mr. Chairman for inviting all of us to be here. I am particularly pleased that General Motors has an opportunity to share some of our perceptions with you, largely identical, certainly similar to those you have already heard for the most part, particularly because of the programs we have in our research laboratories that have, for over the past 20 years, been focusing very heavily on health effects related to air pollution. With me today is Dr. Elaine Wright, who heads our pulmonary toxicity research programs at the current time.

Mr. Chairman, we are convinced that the primary factor that needs to be considered in developing policies for air pollution control and abatement should be peer-reviewed medical literature or studies in particular in the subjects that address the key issues that we have heard talked about today on potential adverse health effects. Our belief is evidenced by our own strong in-house effects research programs and by our large support of extra-mural research programs as well as extensive collegial associations with both government and other industrial laboratories and scientists.

In addition to concerns about the effectiveness and economic impacts which we heard a little about today with various control strategies, environmental health issues have an emotional component. As a physician as well as a researcher with four children, I understand that emotional component.

Public concern stimulates interest and provides incentives to bring policymakers and scientists together to identify the important problems in need of attention, but the public needs accurate, balanced, objective peer-reviewed medical literature in order to do that—information that balances both the known and the suspected potential worrisome risks and the uncertainties of the science. We believe that it is important for the scientific data to be evaluated carefully and objectively.

According to the EPA's most recent air quality data, there are 96 areas in the country that exceed the health-based standard of 0.12 parts per million for ozone. The vast majority of these areas have recorded maximum levels at or below 0.20 parts per million with many areas being marginally above the standard, for only a few hours, over the 3-year statutory compliance period.

This is important in that, our own research and the published research of other laboratories continues to show that responses to low levels of ozone at concentrations at or near the current standard of 0.12 are small, reversible, and generally induced only when the subjects are heavily exercised. This is not to say that such exposures are acceptable.

General Motors recognizes that there is a statutory and medical ethical obligation to comply with the requirements of the Clean Air Act, and we urge that. We urge that all areas of the country including those that we just heard referenced earlier, where there are regionally important problems, come into compliance with the health-based standards for ozone and carbon monoxide that cur-

rently exist. However, our findings do suggest that, contrary to what is being represented frequently in the media and elsewhere today, the current noncompliance situation does not constitute a new public health crisis.

Most importantly, General Motors believes that the standard setting process that is used by the EPA in peer review, expert panels and highly focused medical discussions, is excellent and is indeed the process that should be used to set standards. We support that process.

The key issue is defining what constitutes an adverse, permanent, or disease-exacerbating respiratory health effect. As we heard, both the American Thoracic Society and the EPA in their recent draft papers have suggested formulas for distinguishing what constitutes adverse health effects and distinguishing them particularly from physiological adjustments which may not signal disease or injury itself.

While there is yet no clear consensus as to what constitutes an adverse health effect—an EPA panel is usually split right down the middle on these definitions—it is true that there is agreement that every measurable effect, as our dosimetry techniques get better and better and our statistical techniques get better and better, that every measurable effect is not necessarily adverse. While effects that are three or four times the current standard which underpin almost all of the data that we have heard today so far discussed are adverse to health, the current standard does protect health.

It has been said that children, the elderly, and individuals with preexisting disease such as asthma, show increased sensitivity to ozone exposure. To the contrary, EPA's review of the existing data set forth in the criteria document, which has been referred to a number of times today, indicate that individuals with preexisting disease respond to ozone in about the same way as do those who are normal healthy individuals. Our own studies in our laboratory confirm that assessment.

With regard to purported adverse health effects in children, epidemiological observations do not always produce consistent results. Dr. Ken Gross, in our laboratories, in collaboration with the American Health Foundation and Ranchos los Amigos Hospital, carried out two large studies continuing into our third year with a total of 350 children. We have studied them as they went about normal daily activities and exercise in California summer camps.

The data from these studies is complex. Analysis is underway. In addition to those epidemiology studies, a number of different laboratories have studied pulmonary function of children under controlled laboratory conditions.

None of the work that has been published, and that which is emerging from our laboratories, supports the claim that the children respond more severely to ozone than adults. We are finding effects in our surveys that are about half the magnitude observed in earlier studies.

Finally, with regard to concerns about long-term effects, existing evidence does not support claims that responses in animals, such as fibrosis or infection, when they are exposed to the very high levels of ozone, above .5 parts per million, are representative of effects of substantially lower concentrations where human exposure occurs.

Our studies are not yet completely in the peer-reviewed literature, particularly the ones that are in my prepared statement that you have on record concerning carbon monoxide, which I will not address at this point, but the results have raised a number of important questions for both ozone and carbon monoxide. Those effects are essentially that, when one looks at real world of ambient concentrations, the medical literature shows precious little evidence that humans do not find themselves protected with a reasonable margin of safety at the current standards.

Senator BAUCUS. Thank you, Dr. Green.

Dr. GREEN. You're welcome.

Senator BAUCUS. Dr. Boushey, we will hear your statement now, please.

STATEMENT OF HOMER BOUSHEY, UNIVERSITY OF CALIFORNIA, SAN FRANCISCO, CA

Dr. BOUSHEY. Thank you, Mr. Chairman.

I am Homer Boushey, a Professor of Medicine at UCSF and a specialist in lung diseases. My research interest for years has been on how the lung responds to inhaled materials.

Let me say at the outset that I think that the Clean Air Act has been very important in improving the quality of air in our most polluted cities in the late 1950s and 1960s. Its reauthorization is very important.

I am going to focus my remarks today on sulfur dioxide, a ubiquitous pollutant and a product of fossil fuel combustion. It is most common in the older industrial cities of the northeast and midwest, but is found wherever oil is refined or coal is burned to produce power, as around Billings, MT or Bakersfield and Long Beach, CA. Sulfur dioxide has some important effects on its own, but its products, the sulfates, also have potential effects on human health and are probably most important for the effects of acid rain on life in the freshwater lakes and waterways.

The first suggestion that the sulfur dioxide group of pollutants adversely affect health came from observations made in the great crises of the fifties. In the most notorious of these in London in 1952, the death rate briefly reached a level that had not been reached since the influenza epidemic of 1919. We do not really know what the mix of pollutants was exactly, because we did not have our current monitoring systems, but SO_2 is estimated to have been about 1.5 parts per million, 10 times the EPA's current standard for the United States of 0.14 as a 24-hour average.

Although many studies showed a relationship between sulfur dioxide and daily mortality rates, laboratory studies of people did not show acute short-term effects of exposure to low levels of SO_2 until 10 years ago when laboratories, my own included, began to expose people with asthma to sulfur dioxide. We found that with as little as 5 minutes of exposure to 0.5 parts per million during mild exercise, people with asthma developed attacks of bronchospasm with symptoms of wheezing, shortness of breath, and chest tightness.

Many of our subjects asked for treatment after this brief exposure, and without treatment, the symptoms persisted for as long as an hour. This work has been repeated now in several laboratories.

Asthma is not a trivial or rare disease. It affects between three and eight percent of the American population. It is the most common chronic disabling disease of children, and is an increasing cause of mortality throughout the developed world. Sulfur dioxide is not a rare pollutant. It is quite widespread in the United States, and even brief exposures can cause asthma attacks in people with the condition.

The existing primary air quality standard for the U.S. is 0.14 parts per million as a 24-hour average. That means that levels may be very much higher than that for 10 or 15 minutes, as long as the levels are otherwise low enough to average out to below 0.14 over the 24 hours.

This consideration led California to adopt a short-term standard of .25 parts per million for one hour. The EPA decided not to establish a similar short-term standard. It has since been discovered that in Rothschild, Wisconsin, asthmatic children in the elementary school complained that they could not exercise in the school yard. The levels of SO_2 in the schoolyard were measured as high as 5 parts per million for 5 or 10 minutes due to out-gassing from a nearby pulp paper mill, even though the 24-hour averages were below 0.14 ppm and thus met the EPA's standard for SO_2.

Weyerhauser, the owner of the mill, agreed to take immediate and long-term steps to reduce SO_2 emissions in response to the community's request, but they were not required to do so. They are a responsible corporation and did this out of their own interest in cooperating with the community, not because air quality standards required them to do so.

The current standards allow high short-term levels of SO_2 that have interfered with children playing in schoolyards in areas around sources of SO_2. In Port Angeles, Washington, monitoring sites have measured SO_2 at over 1 part per million, and the Port Angeles Community Hospital is located between the monitoring site and the source of the SO_2.

Sulfur dioxide is rapidly dispersed and converted to sulfates, and, as you have heard this morning, there is concern over the potential effects of acid aerosol on human health. A study conducted in Southern Ontario showed that the levels of sulfates correlated with visits to hospitals for respiratory complaints.

In summary, Mr. Chairman, sulfur dioxide and its products are common and widespread pollutants, but their effects on human health are not uniformly felt throughout the population. They are most felt by people with asthma and perhaps other lung diseases, a group that I believe should be protected and that I believe the Clean Air Act of 1970 intended to protect.

Thank you for allowing me to testify.

Senator BAUCUS. Thank you all very much.

We have a vote in progress, so the subcommittee will have to recess for approximately 10 minutes.

[Recess.]

Senator BAUCUS. Subcommittee will resume its sitting.

Dr. Robert Phalen is our next panelist.

STATEMENT OF ROBERT PHALEN, UNIVERSITY OF CALIFORNIA, IRVINE, CA

Dr. PHALEN. I am going to skip some of my introduction in the interest of time, Mr. Chairman. I am a research toxicologist with a specialty in injury to the lung. Additionally, I served for 8 years as chairman of our University Medical Center's Ethics Committee for Human Studies, so I have reviewed about 2,000 protocols of human research. That has given me a very great familiarity with the application of animal data to humans.

My personal research involves computer modeling and animal studies on mixed air pollutants. I would like to touch on four topics that are drawn from my scientific experience.

Mr. Chairman, I appreciated your comment about the number of breaths we take. If you add everything up, we eat and drink a couple of quarts of food and water each day, but in the same period, we can breathe about 20,000 quarts of air. So the potential for exposure to environmental air contamination is large.

The first topic relates to the type of toxicity we see with ozone. There has been some questioning about whether or not the type of effects that we see are really serious or only mild and transient.

We have been told that ozone penetrates deeply to the very delicate regions of the lung, and numerous studies involving ozone exposure of laboratory animals which are very good models for people cause us great concern. They are not trivial types of events.

Number one, we see the killing of critical cells in the lung, the death of major cell populations. We see interference with the ability of the lung to keep itself clean. This is quite alarming to a toxicologist. We see a decrease in the lung's ability to kill microorganisms, which predisposes to infection. We see impairment of the immune system, another important defense system. And we see a loss of integrity of the tissues of the lung, so contaminants of all types can soak into the body more effectively after ozone exposure.

Although these effects resolve and could be said to be transient, during the period of injury, one is predisposed to exposure from environmental carcinogens, environmental infections, and so on. Although the acute response to ozone goes away, there is a period of vulnerability that can lead to very unacceptable consequences.

The second topic involves safety factors. In the U.S. we have an enviable record for protecting public health through the cleanliness of foods, medications, and the environment. Our protection has not arisen from ignoring effects seen in the laboratory or from assuming thresholds will occur at low concentrations. Our protection of public health has largely been due to the application of adequate safety factors.

As I mentioned in my book, "Inhalation Studies," when one takes the smallest accepted safety factor in most areas of safety evaluation still, one can have adverse effects in say three percent of the population. That level of adverse effect is very consistent with what we are hearing about air pollutants possibly being responsible for 5 to 10 percent of the deaths in the country, which certainly would account for the 50,000 number mentioned earlier. Clearly, with ozone we can not maintain a safety factor of even 10. We are lucky to have one of two.

The third topic is children. We have been told about children. They have immature lungs when they are born. This is evidenced by the fact that during the first year of life, kids have about 12 respiratory tract infections, whereas adults have only 2 to 3 per year. Their defenses are really poor. They have to breathe as a newborn, about three times the air per pound of body weight as an adult, their airways are smaller, and they trap pollutants more efficiently than adults. I think if we really wanted to adequately protect children—and this is probably economically impractical—but we would have a safety factor of 5 to 10 just for them.

The last topic is mixtures. There is confusion over mixtures of pollutants. Not all mixtures are more toxic than what one would expect by adding the single contaminants. In our laboratory studies, we have seen some alarming interactions of ozone and oxides of nitrogen. They form acids as well, and along with sulfuric acids, there is evidence that they interact with ozone.

In conclusion, there is no doubt that populations in the United States are experiencing adverse effects from air pollutants. There is a health crisis to that extent, from existing air pollutants. Ozone is probably the most toxic of all these contaminants.

Our current standards do not give us the traditional safety margin that we have had in this country. It would be extremely unwise to relax our compliance, and I personally think it would be wise to consider tightening ozone standards if we can.

Thank you.

Senator BAUCUS. Thank you very much, Dr. Phalen.

Next we will hear from Mr. Michael Schonbrun.

STATEMENT OF MICHAEL SCHONBRUN, NATIONAL JEWISH CENTER FOR IMMUNOLOGY AND RESPIRATORY MEDICINE, DENVER, CO

Mr. SCHONBRUN. Mr. Chairman, I want to thank you and the committee for giving me the opportunity to appear before you today. My testimony will concern the health effects of air pollution, particularly carbon monoxide in Denver, Colorado, and will, I hope, provide some human dimension to the issues we are discussing here today.

For the past 7 years, I have been President and Chief Executive Officer of the National Jewish Center for Immunology and Respiratory Medicine, which is the only research medical center in the United States that focuses all of its efforts on lung disease and immune system disorders. I have a bachelor's degree in political science from Yale and a law degree from the University of Pennsylvania and 18 years' experience in health policy matters. Unlike my fellow panel members this morning, I am neither a doctor nor a biomedical scientist.

Patients, both adult and pediatric, come to the facilities of National Jewish from all 50 States and many foreign countries. Most are sent by physicians. It is no small irony then that a research center such as this one, specializing in lung disease, is located in one of America's most polluted cities. It does, however, make for a very interesting real-life laboratory.

The past 3½ years, I have also been the chairman of the Denver Metropolitan Air Quality Council. That group is the lead planning agency for the six-county region that comprises greater Denver. It is composed of leaders from the public and private sectors, 20 in all. When then Governor Lamm appointed it in 1985, he instructed us to concentrate on Denver's carbon monoxide problem, which was at that time the worst in the United States out of 305 urban communities.

On those days when Denver is suffering through an especially bad episode of air pollution, the patients at National Jewish call us to complain. They tell their nurses, doctors, and the hospital's administration. Some of our outpatients call to cancel their clinic appointments because they are simply not felling well enough to travel.

The complaints specifically center on tightness in the chest, dizziness, and headache. I want to again reemphasize that virtually all of our patients have something wrong with their lungs. Many of our patients have asthma. About 10 percent have emphysema, and it must be noted that these patients are indeed very tough and very sensitive cases.

Another noteworthy occurrence happens on high pollution days at National Jewish, and here I reflect back on some of the earlier discussion this morning. Our doctors tell our patients on those days, "Do not exercise outdoors today." The committee needs to know that exercise and physical fitness are key components in the treatment regimen at National Jewish, so this advice is given very reluctantly indeed.

Another key fact must be brought to your attention this morning and it is this: the Metropolitan Denver area has, according to a report produced by and statistically validated by the U.S. Department of Health and Human Services, an age-adjusted death rate from asthma, emphysema, and other chronic lung disease, that is 30 percent worse than the national norm and is in the worst 10 percent of the country. There are several possible explanations for this troubling statistic, but at a 3-day conference held two summers ago in Keystone, Colorado, a group of nationally prominent epidemiologists, toxicologists, and pulmonologists determined that air pollution could well be a significant factor in its alarmingly high statistic.

In addition, a monograph also referenced earlier this morning, published in 1985 by the National Academy of Sciences, featured a map that showed possible hot spots in the United States for air pollution-triggered lung disease. Denver was one of the dozen or so areas highlighted.

Now that I have represented these facts to you, I need to say a few words about the type of pollution we are confronted with in Denver. During the past 20 years, the Metropolitan Denver area has had compliance problems with particulates, oxides of nitrogen, and ozone, as well as carbon monoxide. Currently and for the past number of years, we have been within acceptable levels for NO_x and appear to be attaining the standard for ozone, especially if it remains at the .12 level.

Health concerns arising from air pollution occur in Denver mostly during the winter months. That is the high season for

carbon monoxide and the Brown Cloud, as we call our fine particulate problem there.

Much work is being done now to study the composition of the Brown Cloud, where it comes from, what its precise components are, how it moves through the metropolitan area, and what exposure to it people actually have. Much is still not known about that. Much better information is available on the specifics of carbon monoxide in Denver.

We know the 75 percent of the CO in Denver comes from automobiles and the number of vehicle miles traveled is rapidly growing in Denver. We know also, importantly for this morning, that CO is at ground level where people will breathe it. This may be somewhat different for the Brown Cloud, which seems to hover somewhat above the ground.

We also know that the highest concentrations of CO are found in the Downtown area where people come in to work and where many of the region's poor live. We know, too, that a zone of high CO pollution stretches beyond the immediate Downtown area, to include areas where the city's hospitals are mostly found, including those patients such as ours that have compromised cardio-pulmonary conditions already.

We do not know everything about the CO pollution, and therefore, we have been calling for the past number of years for additional research into the health effects of air pollution at altitude, where there is 18 percent less oxygen than at sea level. We also need to know more about the health effects of air pollution on people who already have underlying lung disease.

This group is substantial, not only in Colorado but throughout the west, because of migration patterns earlier in this century—people who flocked to the then clean air of the Rockies for the miracle cure for their lung diseases. These are topics that I believe have received inadequate attention to date, but I do believe that the current information we now have available to us, the epidemiological data, the laboratory data, including recent studies commissioned by the Health Effects Institute, and real-life case studies from National Jewish that I referred to earlier, tell us that air pollution in general and carbon monoxide in particular can be dangerous to human health at levels found in America's urban areas. Aggressive action needs to be taken.

As chronic lung disease continues to be one of the top five causes of death from disease in the United States, and the fastest rising among our most common diseases, up over 300 percent on an age-adjusted basis since 1950, versus a decline of 36 percent for all other causes, we see that there is clearly a national need to accelerate scientific investigation and clinical research into the cause, effects, treatments, and cures of those diseases, particularly because they should be so preventable. As we contemplate spending additional billions of dollars in individual communities to clean up the air, we may need to prioritize our targets. Health effects should be central to such a prioritization effort.

Without being overly parochial, I would like to state that National Jewish is proposing to establish a new research center to focus on environmental disease in order to get a better handle on these

issues. We are now seeking support from public and private sources and are hopeful that this effort can go forward.

I appreciate the committee's attention this morning, and would be happy to answer any questions.

Senator BAUCUS. Thank you very much.

Our final witness is Dr. McFadden.

STATEMENT OF E. REGIS McFADDEN, UNIVERSITY HOSPITALS OF CLEVELAND, CLEVELAND, OH, AT THE REQUEST OF THE AMERICAN MINING CONGRESS AND THE NATIONAL COAL ASSOCIATION

Dr. MCFADDEN. Thank you, Mr. Chairman.

I am Regis McFadden, professor of medicine at Case Western Reserve University in Ohio. I am a pulmonary specialist and I direct the Airway Disease Center at that university and a specialized center of research funded by the National Institutes of Health interested in airway diseases, particularly asthma and Chronic Obstructive Lung Disease. My research interest is in the patho-physiology and pathogenesis of asthma.

I am vitally concerned about atmospheric pollution and am anxious to protect my family and patients from any potential harm from this source. Hence, I fully support the need for environmental regulation and share the concerns of this subcommittee. At the same time, I believe the environmental laws should be based on good science and that additional air quality controls entailing great cost should not be imposed where, on the basis of scientific data, they do not appear to be justified to protect the public health or welfare. I would also like to state that, although I oppose Dr. Boushey on this issue, he and I are friends.

The principal concern about the effects of sulfur dioxide on asthmatics is that individuals, as you have heard this morning, with exquisite airway disease or exquisite airway sensitivity, will suffer adverse effects from brief exposure to this gas at the peak concentrations that occur sporadically around power plants and similar sources. I would point out to you that this concern is not based on any direct observations of this phenomenon, but rather on a series of laboratory experiments that have reported airway narrowing in asthmatics exposed to SO_2 for brief periods.

Questions can be raised about the methods and statistics used in many of these studies, so the clinical relevance of this data is far from clear. Rather than dwelling on such problems, however, I think it more useful to focus on the overall significance of the reported effects.

First, it is important to appreciate that at the concentrations typically involved in these studies, effects have been observed only when the asthmatic's exposure to SO_2 is combined with moderate to heavy exercise or a similar increase in breathing rate. However, even in such circumstances, not all asthmatics are sensitive to sulfur dioxide.

In those who are sensitive, the responses are modest in extent, short-lived, and completely reversible. In fact, they are quite similar to the types of reactions that asthmatics experience routinely in their daily lives in response to many common stimuli. It is impor-

tant for the committee to appreciate that shoveling snow, for instance, walking briskly on a cold day, laughing vigorously, sighing, or even going to bed and to sleep will produce changes in lung function equal to or greater than those observed with sulfur dioxide at the concentrations of concern.

The pulmonary function changes typically observed in these studies are on the order of a 100- to 200-percent increase in specific airway resistance or a 15- to 20-percent reduction in the amount of air expelled from the lungs in one second. Such changes do not show large effects.

By way of comparison, asthmatics presenting to emergency rooms for therapy of acute exacerbations of their illnesses often have decreases in FEV1 of 65 to 70 percent from normal and increases in specific resistance on the order of 900 to 1,000 percent—10 times greater. In contrast to other stimuli such as viruses, antigens, and occupational pollutants, sulfur dioxide does not lead to acute asthma attacks that require emergency therapy or hospitalization for relief.

The symptoms that occur, if any, usually rapidly resolve spontaneously or do so with the use of routine inhaled medications. Moreover, this gas does not make asthmatic airways more sensitive to other triggers of asthma, nor does it appear to have any cumulative or long-term effects.

Thus, sulfur dioxide has none of the long-term consequences described by Dr. Bromberg this morning for ozone. The studies also demonstrate that the use of routine asthma medication, such as a beta-agonist, will protect against the effects of sulfur dioxide.

It should be emphasized that in order for most asthmatics to engage in strenuous physical exertion without getting exercise-induced bronchospasm, they must treat themselves with medication, such as a beta-agonist, either before or after the exercise. Since sulfur dioxide produces responses in asthmatics only in conjunction with moderate to heavy exercise, the use of this medication will take away the effects of the sulfur dioxide.

Even if the arguments and the literature regarding the effects of sulfur dioxide are accepted at face value, examination of what is required to produce a response in the real world points to the unlikeliness of that occurrence. The available data indicate that in order to experience broncho-constriction, an asthmatic must be exercising out of doors at a moderate or higher level, must be off medication, must be sensitive to sulfur dioxide, and must be exposed to a peak concentration of around 0.4 or 0.5 parts per million or higher for a period of at least 2 to 5 minutes.

EPA has estimated that with current air quality, the combination of such peak SO_2 exposures and exercise would occur in only about 1 percent of asthmatics, once per year, and only a further fraction of those exposed would experience a response. In the context of all the day-to-day influences producing similar effects in asthmatics, the occurrences of one or two additional transient responses due to SO_2 in a relatively few asthmatics would not have any appreciable impact on their health or activity patterns or pose a threat to the public health. It is thus not surprising that there is no epidemiological or other real-world evidence of a relationship

between sulfur dioxide at current ambient levels and adverse health consequences in asthmatics in the general population.

In short, I do not think that these data provide a justifiable basis for additional controls for sulfur dioxide. Thank you.

Senator BAUCUS. Thank you very much.

Dr. Boushey, you mentioned that you thought the Clean Air Act was designed to protect the sensitive populations, and I want to tell you that is true. That is the purpose of the Clean Air Act.

In fact, I think it is important to know that Congress emphasized that the standards in the act should protect, "Particularly sensitive citizens, such as bronchial asthmatics and emphysematics, who in the normal course of daily activity are exposed to the ambient environment." That is in the legislative history of the act.

The standard is statutorily sufficient whenever there is, "An absence of adverse effect on the health of a statistically related sample of persons in sensitive groups from exposure to ambient air." It is quite clear that the standards in the act are designed to protect not only healthy people, but also significant populations of sensitive people.

With that in mind, I would like your comment, if you would, on Dr. McFadden's points, particularly whether you believe there should be a shorter standard for SO_2 emissions. I say that in part, because in my home State of Montana, particularly Billings, MT, an asthmatic woman died during a high SO_2 exceedance. I don't know whether she was vigorously exercising or not, frankly. But it is documented that there was a high exceedance at that time, she was an asthmatic, and she did die.

In trying to find the truth here and getting to the heart of the matter, I would appreciate it if you could comment on those points.

Dr. BOUSHEY. I have no knowledge of that particular case, but I can answer generally.

First of all, there is agreement on the observation that short-term exposures of people with asthma to sulfur dioxide while they exercise causes narrowing of the airways and symptoms associated with an asthmatic attack. The problem is with the interpretation of this observation, and I would like to rebut, if I may, some of the points that Dr. McFadden made.

One of Dr. McFadden's points is that there is no real-world observation to support the claim that sulfur dioxide exposure worsens asthma. This is refuted by the events in the schoolyard of Rothschild Elementary School, where the principal, who kept the students' medications in his office, discovered that on days when the paper mill was emitting pollutants, students asked for their medication much more often than usual. His inquiries led to a series of studies conducted by the EPA, documenting that there were transient high amounts of SO_2 in the air of the schoolyard.

These were children who were taking their medications, but who were nonetheless having attacks attributable to sulfur dioxide in the air. So there is a real-world correlation to what I think was easily predictable from consistent laboratory observations.

The attacks are described as reversible, and Dr. Green suggested in his testimony that irreversible permanent changes should be considered an adverse health effect. Asthma is by definition reversible, unless it leads to death. It is a reversible airway disease. But

people who suffer from intermittent reversible wheezing, chest tightness, and shortness of breath, would certainly describe those symptoms as adverse. They feel limited by them, and I agree with them that asthma is an adverse health effect.

The other assertion is that the spontaneous changes are greater than those caused by SO_2. This was not our observation in the people with mild asthma, those who are well enough to take part in a laboratory study.

When they exercised without SO_2, our subjects had very modest changes in their airway caliber. When we added SO_2, they sometimes could not complete the protocol because of shortness of breath and wheezing. They sometimes stopped to ask for treatment.

More severe asthma is indeed a disease associated with spontaneous changes in airway caliber unrelated to SO_2. The argument here is not that SO_2 is the only cause of attacks, but that SO_2 can cause attacks in people with asthma.

Senator BAUCUS. Could you describe an asthma attack?

Dr. BOUSHEY. Asthma is due to narrowing of the airways in the lungs. It comes on often after viral infection, exposure to allergens, or on breathing polluted air.

The narrowing of the airways causes symptoms of tightness of the chest, as if a person can not get a deep enough breath of air, shortness of breath, and wheezing.

Senator BAUCUS. I have never experienced one. Does that mean one experiences significant discomfort or pain?

Dr. BOUSHEY. It is not painful; the symptom is one of shortness of breath. You can feel short of breath without hurting, and it can be an unpleasant sensation.

Imagine how you would feel after running 400 meters. You would feel very short of breath. That shortness of breath would be appropriate for the situation. If you felt that way after walking up one flight of stairs, you would feel that was distressed. Shortness of breath, tightness of the chest, and wheezing are the symptoms of an asthmatic attack. Some patients describe the sensation as one of "air hunger."

Senator BAUCUS. Do either of you think that in order to comply with the act, that medication is sufficient? Should we set the precedent that there is an adequate margin of safety so long as there is medication available?

Dr. BOUSHEY. I would be made very uncomfortable by that as a precedent, that we should permit air pollution because people can take medication to protect themselves. Incidentally, my colleagues and I did research in my laboratory on the effects of standard doses of asthma medication on SO_2-induced attacks and they were not protective.

Even giving them 10 times the usual dose was not fully protective and did not normalize the response to SO_2. It takes a lot of medication, I believe, and I disagree with the precedent that we should accept air quality because some people can take medication and thereby tolerate it.

Senator BAUCUS. Dr. McFadden.

Dr. MCFADDEN. I would agree with that. I do not think we ought to set a precedent.

This is probably not the forum to quarrel with Dr. Boushey, although I enjoy doing that, but I would point out two things. The first is that being exposed to SO_2 during exercise is not having a young child or an adult asthmatic exercise by running through a field of pollen to which he is sensitive. The latter will put him in the hospital for four or five days.

Exposure to SO_2 may make him short of breath and may make him have an asthma attack, but that will last, if no medication is given, on the order of perhaps an hour. It is not like taking an aspirin for an aspirin-sensitive asthmatic. One single aspirin, or one-tenth of one single aspirin, can put someone like that into the hospital for a week.

So we are not talking about the same sized effect. I do not deny at all that there is an effect on exposure to SO_2 with asthmatics. My argument is that, that effect although there, does not have the same consequences as effects to other sorts of things to which people are exposed.

Senator BAUCUS. Senator Lieberman.

Senator LIEBERMAN. Thank you, Mr. Chairman.

If I may just clarify, Dr. McFadden, are you suggesting that there ought not to be a health-based standard in the Clean Air Act?

Dr. MCFADDEN. No, not at all. I am suggesting that the current standards for SO_2 may be adequate. That is all I am suggesting, sir.

Senator LIEBERMAN. Because of the availability of medication to deal with problems beyond that?

Dr. MCFADDEN. I would agree with Dr. Boushey about that paint. It would be a bad precedent to say that we could control the effects of air pollution by giving people medication or put that idea into legislation.

On the other hand, as I stated in my opening comments, the use of medication by asthmatics is relevant to the SO_2 issue. Regardless of the presence of SO_2 in the air, most asthmatics must use medication in order to prevent or ameliorate the bronchoconstriction caused by vigorous exercise alone, and since SO_2 produces effects in asthmatics only in conjunction with such exercise, the use of the medication to deal with the exercise will necessarily also prevent or ameliorate any effects of any SO_2 that is encountered during the exercise.

It is just that the effects of SO_2 are not very large in my view. It is important to appreciate that the studies that are in the literature are designed to minimize the effect of exercise per se on the asthmatics. The way that is done is simply increasing the humidity, and the temperature of the air that the asthmatic is breathing. You can then add in whatever pollutant you want. We are not looking at exercise in the real world, which in my experience, far outweighs the effects of any SO_2 that is added to it.

Senator LIEBERMAN. Dr. Green, correct me if I am wrong, but I heard you suggest earlier that you believe exposure to ozone does not lead to permanent lung damage, but rather leads to transitory experiences of lung dysfunction. Is that correct?

Dr. GREEN. Yes, that is right. I was referring to the rather large data base that relates to animal studies in which rather large con-

centrations, up to 0.5 have in fact found effects which revert or become adaptable after returns to clean air.

Senator LIEBERMAN. This is contrary to what I have been reading and hearing certainly in the 4 months that I have been in the Senate. There was a recent Dutch/American conference—I don't know if you are familiar with it—from which I saw some reports that suggested growing evidence of permanent lung damage, based on animal studies. The Acting Administrator for Air of EPA testified in late February before a House committee to the same effect. So I am puzzled by what you are telling us and I invite you to say some more.

Dr. GREEN. Sure. Let me comment very briefly and then ask Dr. Wright, who is very familiar with that data, to comment in an additional minute.

We find that the information that you are referring to is confusing and there has been recent material that has appeared in the press that would indicate otherwise, but by and large, the evidence that we are aware of that has been published does not support that. That includes the series of six or eight papers that have been presented in the past month, the Health Effects Institute's programs and the EPA's programs.

Elaine, you may want to comment more on that.

Dr. WRIGHT. You may be referring to the studies performed by the EPA Health Effects Research Laboratory scientists on the chronic exposure of rats to what they termed an ambient pattern of ozone where they had daily peaks of .25 parts per million. In fact, we have similar studies that were designed somewhat differently but have been presented side by side with those EPA studies.

We are in the same scientific fora with those scientists and we have found that we essentially agree, in spite of the design differences in the studies, that 18 months, which is a large percentage of a rat's lifetime, of exposure to levels of ozone far exceeding the current standard, do not cause permanent, irreversible damage to the lung. There are changes in pulmonary function in these animals that revert.

There are some morphologic changes that parallel the reversion of the functional changes that would be termed fibrosis. However, the interstitial fibrosis does in fact resolve with time. There are some irreversible effects and we are simply not sure what the clinical significance or the functional significance of these changes are, but our studies do not disagree with those studies.

Senator LIEBERMAN. Dr. Phalen, I take it that you would disagree with the notion that exposure to ozone does not cause permanent lung damage?

Dr. PHALEN. Yes, I would disagree with that, even if some of the responses were transient, which they are. For example, a reduction in the ability to fight off infection. So that lasts only a week or two? If you get an infection, that is a drain on your bank account of health.

A temporary reduction in the permeability of the body? Every breath we take has tens of thousands of particles in it, and we have to exclude those from our bloodstream and from our tissues. If that exclusion barrier breaks down, even temporarily, we load our bodies up with other contaminants. Even the temporary effects are

alarming because they represents a decrease in one's ability to fend off carcinogens and infectious agents.

But I also think that the types of changes that are seen in long-term studies imply that there is an irreparable component of ozone damage. Your lungs are very similar to your skin. Let's take sunburn, for instance. If you get a severe sunburn, 2 weeks later your skin appears normal. It is reversible. But we also know that people who have that repeated exposure or chronic exposure are susceptible to a whole variety of skin diseases, including skin cancer.

Senator LIEBERMAN. Thank you, Mr. Chairman.

Senator BAUCUS. Dr. Green, the studies you and your associate referred to—are those published studies?

Dr. GREEN. Yes, they are. Our studies that we referred to have been published.

Senator BAUCUS. And also the ones Dr. Wright referred to?

Dr. WRIGHT. Yes, that is correct. The EPA studies have not yet been published in the peer-review literature. They have been presented publicly.

Senator BAUCUS. I am talking about your own in-house studies.

Dr. WRIGHT. Our own in-house studies have been published, yes.

Senator BAUCUS. Okay.

I wonder, Dr. Phalen, could you also indicate if the studies you are referring to that have a contrary assertion are published?

Dr. PHALEN. I have a list of relevant tables from an excellent summary document that I will give to your staff.[1]

Senator BAUCUS. All right.

Dr. Green, earlier we heard Dr. Lippmann say, in response to my question of the degree to which ozone effects are reversible that a lot of it is not reversible and that some of it is permanent and chronic.

I wondered if I could have Dr. Lippmann come to the table and indicate his views.

Dr. LIPPMANN. We are clearly going on limited information at this point. Putting it all together, and having reviewed the entire literature base for ozone for review papers that I have presented, it seems to me compelling that there is some incremental damage from each episode of exposure. It seems to me that the objective evidence is consistent between the EPA studies and the GM studies, but we have to put in several caveats in interpreting what they may mean to humans.

One thing that is known but is not taken into account is the difference in dosimetry between the rat and humans. I indicated that the rat gets at least twice as much ozone to that target zone as the humans, and furthermore, the rats are exposed at rest.

The rats in these studies are exposed primarily in the daytime. They are a nocturnal animal and not very active during the day, and they can not move around much in a cage.

Human responses would be even more if you allow normal activity to intervene. So when the EPA simulated the real concentration,

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[1] From "Air Quality for Ozone and Other Photochemical Oxidants," Volume I, section 1.8, pp. 1-103 to 1-136, U.S. EPA-600/8-84-020aF, 1986.
The above cited publication has been received and is retained in committee files.

they were underestimating the challenge that humans would get because of the difference in dosimetry.

Secondly, the humans are exposed to ozone with acid aerosols and other pollutants, and we have a lot of evidence that shows that potentiates the response to ozone. Finally, it is clear that in both the EPA and GM studies that, after 18 months of exposure and 6 months of follow-up, a lot of the changes in functional structure have reversed. This may be immaterial because we do not get exposed for 20 years and then have clean air thereafter.

During the course of the exposures, the structural and functional effects were progressing. They did reverse when exposure stopped, but that is not an appropriate model because exposure never stops.

Senator BAUCUS. Thank you.

Equal time, Dr. Green?

Dr. GREEN. I find myself in a great deal of association with the remarks that Dr. Lippmann has made. He is pointing up in the last part of his comment something that is really very important. It relates to the dosimetry, that is the amount of the ozone that people are breathing over an integrated period of time, whether several hours that a standard is found to be in exceedance, or whether it be several days that a standard is found to be in exceedance.

That underscores why many of us believe that the actually important thing to consider for the future is whether or not the current standards adequately represent that amount of integrated ozone or other pollutants over a period of time. While we say that our studies in our laboratories, as Dr. Lippmann has pointed out, are in essential agreement with others, the issue here is whether or not in the real world, an occasional exceedance for a short period of time is adequately or fairly being monitored or measured by the form of the current standard. I would applaud the idea of trying to do a better job of measuring the actual amount of dose.

Senator BAUCUS. Is it a question of a longer standard?

Dr. GREEN. I am not sure what you mean by longer. I think that the issue really is in terms of the terminology that is being used today of finding a robust standard. That is, to find one that statistically, fairly measures and integrates the actual amount of ozone or other pollutants over a period of time that people are actually being exposed, whatever that may be.

I personally do not know whether that would mean it would be a longer or shorter standard. I just don't know.

Senator BAUCUS. Does General Motors support the reauthorization of the Clean Air Act legislation that would bring reductions in emissions for motor vehicles?

Dr. GREEN. I simply do not know the answer to that. I can talk a great deal about our health effects and whether or not our emissions people have a view that should be formulated more precisely, I simply am not aware.

Senator BAUCUS. Has anyone said anything so outrageous that it deserves a response today?

None whatsoever.

Senator Lieberman.

Senator LIEBERMAN. Just one last question of Dr. Green.

I believe that earlier you testified that there could be lung dysfunction cases of heavy exercise. Would that include children at play on a hot summer day?

Dr. GREEN. It depends on the amount of exercise and how one measures it. For example, our studies with children in the summer camps, along with Dr. Lippmann's, are now emerging and the differences between them are not really frankly very large. The actual amount of distress is somewhat smaller in terms of the measured effects.

I think that it is more important to be aware of the fact, as many people have, that when one measures people in a chamber, one can tell exactly what is being given to them for the experiments, but one can not tell whether or not in the real world, when a summer camp study, for example, does or does not show an effect that, that may be caused by one particular pollutant or another.

Senator LIEBERMAN. So do you advise your children not to play aggressively on a hot summer day?

Dr. GREEN. I would have a tough time with my kids if I tried that. All four of them are very athletically inclined.

I do in fact give them the same advice that I would suspect every physician on this panel would. I do not tell my children to reduce exercise heroically unless the ozone level is considerably above the current standard because I do not see any effects on them when it is in the area of compliance.

I certainly do ask that they pay close attention when the weather is getting bad, particularly when it is hot and when there might be other things in addition to ozone where they might be traveling or playing, and that they listen to their own bodies and pay attention to the way that they feel.

Senator LIEBERMAN. There might be other pollutants in the air, you mean, besides the ozone?

Dr. GREEN. Possibly.

We have heard that discussed today in a very appropriate way, particularly with the possibility of acid aerosols confounding the picture somewhat.

Senator LIEBERMAN. Thank you, Mr. Chairman.

Senator BAUCUS. Thank you all very much. You have been most helpful.

The subcommittee is adjourned.

[Whereupon, at 1:10 p.m., the subcommittee was adjourned, to reconvene at the call of the Chair.]

[Statements submitted for the record follow:]

Testimony of Philip A. Bromberg, M.D.
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Submitted to the United States Senate Subcommittee on Environmental Protection, of the Committee on Environment and Public Works

April 18 1989. Washington, D.C.

Mr. Chairman and members of the subcommittee: Thank you for inviting me to testify before you on issues relating to health effects of ambient ozone and carbon monoxide. I am Dr. Philip Bromberg, Professor of Medicine, Director of the Division of Pulmonary Medicine and Director of the Center for Environmental Medicine at the University of North Carolina School of Medicine in Chapel Hill, N.C. Over the past 12 years I and other members of the Center for Environmental Medicine have conducted experiments designed to evaluate health effects of exposure of normal volunteers and patients to several air pollutants including ozone and carbon monoxide. These studies have been performed in collaboration with scientists from the EPA Clinical Studies Branch in Chapel Hill.

Ozone:

We are all aware of widespread non-attainment of the national ambient air quality standard for ozone (O_3) which is presently set at 0.12 ppm for a one hour period. In discussing the effects of ambient O_3 pollution, I will confine my remarks to human health issues and will not comment on deleterious effects of ambient ozone on crops, on vegetation, and on materials, although these adverse effects also merit serious consideration.

I first wish to point out that ozone is a highly reactive gas. More than 90% of the ozone inhaled by humans experimentally exposed to low levels of this gas never sees the light of day again. It is fixed to the respiratory tissues by chemical combination.

It is well established from studies of groups of normal adults and children, both in environmental chamber experiments and in exposures to ambient air, that exposure to ozone levels as low as 0.12 ppm causes significant symptoms (especially cough on inspiration) and reduction in lung function. (McDonnell et al, 1983, 1985). In 2 hour exposures in environmental chambers the subjects intermittently performed vigorous exercise, sufficient to raise ventilation to high levels for an aggregate of 60 mins. The results of all such published studies (to 1986) have been analyzed by Mazucha (1987).

In longer exposures (6 3/4 hours) to only 0.12 ppm ozone, even more striking symptoms and decreases in lung function have been observed despite the use of only moderate levels of exercise during the exposure. Recent studies in EPA's Clinical Research Branch laboratory have found adverse effects on lung function of 6 3/4 hour exposures to ozone levels of 0.10 and 0.08 ppm, levels that are even below the current 1-hour standard.

Individuals appear to differ widely in their sensitivity to ozone. Thus, in a group of 10 apparently comparable healthy individuals exposed in environmental chambers, one or two may have no symptoms or changes in lung function, one or two may have marked symptoms and striking decreases in vital capacity, and the others fall between these two extremes. The reasons for this variation in individual sensitivity remain obscure, but a significant fraction of normal individuals display high sensitivity to ozone.

The National Ambient Air Quality Standards are designed to protect not only healthy individuals but also subpopulations which may exhibit exaggerated vulnerability to an environmental toxicant. Evidence from field studies of asthmatics in Los Angeles (Whittemore and Korn, 1980) and in Houston (Holguin et al, 1985), and from a large scale population study in southern Ontario (Bates and Sizto, 1983, 1987) suggests that increased ambient air ozone levels are associated with increased respiratory symptoms and medical visits, especially

involving asthmatic patients. However, several experimental short-term acute exposures of asthmatic subjects to ozone in environmental chambers have failed to reveal any remarkable sensitivity of these patients to ozone. (Linn et al, 1978; Silverman, 1979; Koenig et al, 1985). Similar experimental exposures of patients with chronic obstructive bronchitis and emphysema (at rather low levels of exercise) have likewise failed to reveal undue sensitivity of this group to ozone. (Solic et al, 1982; Hackney et al, 1983; Kehrl et al, 1983, 1985; Linn et al 1982, 1983; Kulle et al, 1984). Finally, work from Dr. Horvath's laboratory on older subjects does not show them to be unduly sensitive. (Drechsler-Parks et al, 1987).

Thus, the evidence from field studies suggesting that patient with respiratory disease are particularly sensitive to ambient ozone pollution does not appear to be supported by the evidence from short-term experimental exposures to ozone of specific patients with common chronic respiratory diseases. Longer experimental exposures, repeated daily exposures and other studies are needed to more fully assess this apparent discrepancy.

Nevertheless, the existing information on normal children and adults indicates that the present air quality standard for ozone of 0.12 ppm ozone for one hour provides little or no margin of safety against the acute effects of ambient air ozone exposure, for at least 10% of the population.

Are these adverse ozone effects — viz, burning or raw sensation in the trachea (windpipe), cough (spontaneous or provoked by deep inspiration) and decreased ability to take a deep breath — merely temporary limitations, albeit harmful ones, or do they herald irreversible impacts on respiratory health?

Unlike the situation for cigarette smoking or certain other toxicants, in which irreversible health effects of repeated exposure are now well appreciated, there is no incontrovertible proof that repeated exposure to ambient "oxidant" type pollution causes either chronic obstructive lung disease or lung cancer.

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These are of course difficult health endpoints to establish in a population in which powerful confounding risk factors like cigarette-smoking are so prevalent. Nevertheless, Detels, et al (1987) in their continuing UCLA population study of chronic obstructive lung have provided some evidence that residence in a high oxidant pollution area may be associated with an excessive rate of decline in lung function in non-smokers.

How else can we assess this question? Three general approaches suggest themselves:

1.) are acute ozone effects rapidly and totally reversible after cessation of exposure?

2.) what cellular mechanisms underlie the acute effects observed in human subjects? Do these mechanisms have the potential to cause irreversible changes, either acting alone, or in concert with other factors?

3.) what happens to animals chronically exposed to ozone?

Firstly, after normal subjects have apparently recovered from an acute ozone exposure that resulted in temporary symptoms and lung function decrements, it is possible to show that more subtle alterations are still present. Thus, the airways remain more sensitive to drugs that cause contraction of airways smooth muscle (Golden et al, 1978). Also, a second exposure to ozone, 24 hours after the first, will produce a larger effect than did the first exposure (Hackney et al, 1977; Farrell et al, 1979; Badi et al, 1985; Folinsbee and Horvath, 1986). Thus, there is at least a 24-hour "memory" of the effects of an acute, short ozone exposure.

Secondly, the respiratory surfaces many hours after ozone exposure continue to show excessive numbers of inflammatory cells (Koren et al, 1989). These cells have powerful enzymatic mechanisms that are capable of causing tissue damage. Certainly one would not wish to knowingly cause repeated bouts of airways inflammation just as one would not wish voluntarily to have repeated sun-

burns. In addition, the respiratory surface epithelium which normally constitutes a very tight barrier separating the respiratory system tissues from the external environment, has been shown to become more porous (i.e., more "leaky") after an acute ozone exposure (Kehri et al, 1987).

Thirdly, animal experiments clearly confirm the inflammatory effects of ozone on the respiratory system. Some investigators have proposed that the inflammation is linked to the increased airways reactivity to bronchoconstrictor drugs seen in animals as well as man following ozone exposure. A variety of toxic effects of ozone inhalation on the surface epithelium have also been well documented in various mammalian species. Ozone is highly reactive and would be expected to react predominantly with the lining epithelial cells of the airways which are of course also the cells of origin for lung cancer. Lung epithelial cells, especially airways ciliated epithelial cells and alveolar Type I epithelial cells, are known to be damaged by ozone exposure.

Chronic exposures of rodents and of primates indicate that ozone can induce fibrotic changes around small airways. Combinations of ozone with other pollutants like aerosols of acid sulfates may be especially potent in this regard. It would obviously be highly undesirable were similar effects to occur in humans repeatedly exposed to ozone.

Finally, recent experiments show that O_3 can react with chemicals present in cigarette smoke to generate products that damage DNA in lung epithelial cell lines in culture (Kozumbo et al, 1989).

Thus, despite the absence of irrefutable, conclusive, epidemiologic proof of irreversible changes in the human respiratory system attributable to ambient ozone exposure, there is substantial reason for serious concern about ozone's potential in this regard.

Because of the solid evidence for acute human respiratory effects at very low ozone levels and the reasons for substantial concern about adverse effects

of repeated ozone exposures outlined above, I conclude that it is prudent from the public health standpoint to continue and indeed amplify our efforts to decrease levels of O_3 commonly found in summertime ambient air. The realization of this policy goal is of course a complex matter.

Carbon Monoxide (CO)

The present national ambient air quality standard for CO is set at 9 ppm averaged over an 8-hour period and 35 ppm over a 1-hour period. Exceedences for CO are common and widespread though perhaps somewhat less than is the case for ozone. It is possible that the ambient air monitoring devices providing this information for CO are not always situated optimally for the detection of the highest levels to which people are being exposed.

Unlike air pollutants like ozone or sulfur dioxide, CO does not damage the lungs even though it is taken up into the blood and excreted from the blood via the lungs. Nor does CO combine in an irreversible manner with tissue components as does ozone. Nevertheless, exposure to CO is well known to be potentially lethal.

Present thinking about the mechanism of toxic effects attributable to low levels of CO focuses on the remarkably high affinity of CO for certain proteins like blood hemoglobin and tissue myoglobin that contain heme groups. These heme proteins are essential parts of the oxygen (O_2) transport system. CO competes with O_2 for binding to these proteins and thus impairs the transport of oxygen to the tissues. Whereas oxygen is further used to support critical chemical reactions in the tissues, CO cannot substitute for O_2 in these roles and therefore can only be removed by excretion through the lungs.

Before discussing the effects of elevated levels of blood carbon monoxide-hemoglobin (COHb), I wish to clarify the quantitative relationship between ambient air CO levels expressed as parts per million (ppm) and the percent of hemoglobin oxygen-binding sites occupied by CO (i.e., % COHb). This is important because health effects research focuses on % COHb levels in blood whereas ambient air quality focusses on the ppm of CO in air. There is a well-understood but somewhat complex relation between these two variables that was described in detail by Coburn, Forster and Kane in 1965.

Several points merit emphasis:

1. Even when breathing air containing no CO, the blood COHb level will normally be 0.5 - 1.0%. This is because a small amount of CO is produced by body metabolism. This endogenously produced CO cannot be further metabolized and must therefore be excreted through the lungs.

2. When the ambient air contains CO, the blood coursing through the lung capillaries picks up CO as well as O_2. Given enough time, the blood COHb level will reach a stable value if the ambient CO is maintained. If the blood in the lung capillaries is at least 90% oxygenated, the steady state % COHb will be about 1/7 of the ambient CO in ppm. For example, breathing air with a CO level of 21 ppm, one would expect to eventually attain a blood COHb level of about 3%.

3. The COHb level in the relatively poorly oxygenated fetal blood will rise above the level observed in the mother's blood because there is less O_2 available to compete with CO for binding to hemoglobin. Thus, with the mother continuing to breathe air with a CO content of 21 ppm, the fetal blood COHb level could eventually rise to the 4 - 5% range although the maternal blood COHb level was only 3%.

4. The expected effect of a given level of ambient air CO on blood COHb is not additive to the effect of personal pollution. Thus, a cigarette smoker with 5% COHb, for example, who is then exposed to 21 ppm ambient CO will not develop any further increase in COHb.

Returning then to displacement of oxygen from hemoglobin by CO, and the consequent reduction in oxygen-carrying capacity of the blood, under what conditions might such changes be deleterious to health, and are there particular subgroups in the population who would be at particularly high risk?

Effects of increased COHb on exercise tolerance in normal individuals

Since decreasing the oxygen supply to a tissue may limit the maximum perfor-

mance of that tissue — e.g., exercising skeletal muscle or heart muscle — investigators have examined the effect of acutely-induced increases in blood levels of COHb, ranging up to at least 20%, on exercise performance of normal individuals. Usually CO gas was administered but in one recent publication (Hirsch et al, 1985) an average COHb level of 6.6% was obtained by having the subjects smoke cigarettes, since cigarette smoke contains high concentrations of CO. As expected, an increased level of COHb reduces maximal exercise performance, increases heart rate during submaximal exercise, causes lactic acid to appear in the blood at lower levels of exercise, and decreases exercise time. However, it does not seem that these effects have been demonstrated at levels of COHb less than 4 or 5%. (Ayres et al, 1970; Ekblom and Huot, 1972; Hirsch et al, 1985; Horvath et al, 1975; Pirnay et al, 1971; Vogel and Gleser, 1972).

The reasons for this apparent inability to show deleterious acute effects on exercise performance in normal subjects at COHb levels less than 4 - 5% are not clear since a priori one would not expect a COHb threshold for these effects. Existing technology may not be sensitive enough to detect very low level effects in groups of subjects.

Chronically elevated COHb levels can cause increased red cell mass (polycythemia) as seen in heavy smokers (Smith and Landaw, 1978; Sagone and Balcerzak, 1975). Again, hematologic effects of very low COHb levels are hard to demonstrate although there is no a priori reason to expect a COHb threshold.

Effects of increased COHb in the presence of hypoxemia or disease

Diseases or circumstances associated with limited or impaired delivery of O_2 are likely to be particularly sensitive to the effects of COHb on O_2 metabolism. These include the hypoxia encountered at high altitude and the hypoxemia of the normal fetus in utero, as well as cardiovascular diseases associated with

limited cardiac output, coronary artery disease, cerebral vascular disease or peripheral arterial disease, hypoxemic lung disease and anemia. Many of these conditions are particularly prevalent among older individuals.

For any given CO pressure, a decreased O_2 pressure will increase the fraction of hemoglobin (or myoglobin) in the COHb (or COMb) form. Thus, to protect unacclimatized visitors to high altitudes, California has promulgated a CO air quality standard more stringent than the national standard.

Effects on the fetus

The fetus is at particularly high risk to the effects of maternal inhalation of CO. In pregnant sheep inhaling CO-enriched air, the steady state levels of COHb attained in the fetal lamb blood were about 50% higher than those found in the maternal blood (Longo and Hill, 1977). This is attributable to the reduced O_2 pressure characteristic of umbilical vein blood returning to the fetus from the placenta.

In humans, maternal tobacco smoking is associated with decreased birth weights at full term and with an increased incidence of various complications of pregnancy, including prematurity, placenta praevia, and abruptio placenta. Similar findings have been reported to result from chronic hypoxic (altitude) hypoxia (McCullough et al, 1977; Moore et al, 1982), and the decrease in birth weight found in newborns of mothers living at high altitude is more profound in smoking mothers (Moore et al, 1982). Thus, CO is likely to be a major contributor to these adverse health effects of maternal cigarette smoking. Cigarette-smoking pregnant women may also be more susceptible to the deleterious effects of other agents, such as ethanol, on fetal growth and development.

The fetal central nervous system is particularly susceptible to CO. Delayed or possibly even long-term impairment of neurological and intellectual development has been suggested to result form maternal smoking during pregnancy (Dept.

of Health, Education and Welfare, 1979; U.S.EPA, 1979; Longo, 1977). Studies in rats of Fechter and his colleagues support the notion that modest levels of maternal CO exposure may find the fetus extraordinarily susceptible to the development of irreversible neural changes (Fechter and Annau, 1977 and 1980; Mactutus and Fechter, 1984 and 1988; Dyer et al, 1979). Fechter (1984) has reviewed the effects of CO exposure on early fetal development.

Effects on the nervous system

Extensive studies in volunteers of the effects of acutely elevated COHb levels on perception and certain motor skills suggest that reproducibly positive findings require a saturation of at least 5%. (For evaluative reviews of this complex area, see Laties, 1980; Laties and Morigan, 1979; Benignus et al, 1983).

Recent studies of hearing loss provide an interesting example of possible enhancement of CO toxicity when a compensatory increase in regional blood flow is inadequate. It has been known for some time that cigarette smoking may be associated with hearing loss (Chung et al, 1982), and permanent hearing loss has been reported in victims of CO poisoning (Morris, 1969). In rats, CO exposure of intermediate severity causes an increase in cochlear (inner ear) blood flow that compensates for the local hypoxia (Fechter et al, 1987). As a result, CO exposure alone in test animals does not impair auditory function permanently except in concentrations high enough to threaten life. Recently, however, CO exposure was shown to potentiate the persistent auditory damage caused by noise (Young et al, 1987). Noise stimulation may render inadequate the compensatory increase in cochlear blood flow induced by CO, thereby making the tissues vulnerable to the deleterious effects of CO-induced anoxia.

Patients with cerebrovascular disease or with retinal or cochlear impairment do not appear to have been studied for possible adverse effects of low levels of COHb.

Effects on the cardiovascular system

Patients with cardiovascular disease, as noted above, would be expected to be particularly sensitive to the further limitation of tissue O_2 supply caused by increased COHb. The reported work in this area has focused on patients with ischemic heart disease.

Earlier investigations, principally by Aronow and his colleagues, appeared to show that individuals with ischemic disease performing exercise experienced a shortened time to the onset of angina, prolonged angina, and ST segment depression at mean COHb saturations between 2% (Aronow, 1981) and 2.9% (Anderson et al, 1973). Aronow's work, however, has been criticized and discounted for a variety of reasons. A multicenter study sponsored by the Health Effects Institute (HEI) (Allred et al, 1988) in which the data from 63 highly selected male subjects with stable exertional angina were pooled, tended to confirm the earlier findings of Aronow et al and Anderson et al. Significant dose-related changes in the reduction of both time to onset of chest pain and development of ischemic ST segment patterns occurred at mean COHb levels of 2.0% and 3.9% measured postexercise. Using a more representative sample of patients with ischemic heart disease, Sheps et al (1987), could find no statistically significant changes in these endpoints, or in ventricular ejection fractions, following acute elevation of COHb from a baseline of 1.6% to a mean level of 3.8%. Using the same protocol, the same investigators did observe significant adverse changes at 5.9% COHb (Adams et al, 1988). Whatever the reason(s) may be for these apparent differences in COHb threshold among the studies, it appears reasonable to infer that a significant fraction of the population with ischemic heart disease is at increased risk for signs of myocardial ischemia at COHb levels somewhat above that reached at equilibration with the present 8-hour NAAQS of 9 ppm or at COHb levels that may be reached prior to equilibration during more intense exposure, particularly indoors or in heavy, slow traffic.

Recently, the importance of silent (painless) myocardial ischemia in patients with coronary artery disease and its relation to sudden death have been recognized (Cohn, 1986; Pepine, 1986; Savage et al, 1987). There have been no studies of possible effects of COHb on this manifestation of coronary artery disease. Nor have studies of the effects of low levels of COHb on exercise tolerance in patients with severe limitation of cardiac output due to cardiomyopathy, valvular heart disease, or pulmonary hypertension been reported.

Chronic elevations of COHb and atherosclerosis

In addition to the acute effects of CO inhalation on cardiovascular performance status and the subacute to chronic effects of maternal CO inhalation on fetal intrauterine growth and development, chronic CO inhalation may be related to atherogenesis and development of significant arteriosclerotic lesions. The evidence for this assertion is largely epidemiologic in nature, and much of it is related to the increased risk for arteriosclerotic disease in cigarette smokers (U.S. Dept. of HHS, 1983). Elevated COHb levels are of course closely related to cigarette smoking. However, the possible role(s) of other components of cigarette smoke are unclear, and a plausible mechanism whereby elevated COHb levels might enhance atherogenesis (perhaps only in susceptible individuals) has not yet been established.

As part of a critical evaluation of automotive emissions as risk factors for the development of atherosclerosis and coronary artery disease, Clarkson (1988) has reviewed epidemologic studies of the relation of human occupational CO exposure to coronary artery disease and experimental studies of the effect on atherogenesis of CO exposure in animal models (pp. 617-9). Occupational CO exposure appears to correlate with increased incidence of coronary artery disease if COHb levels exceed about 5%. Clarkson summarizes the animal data as tending to show "that CO probably affects atherogenesis only when coadministered

with a moderately atherogenic diet, and that the coronary arteries are more affected than other arteries."

In conclusion, I should like to point out that important questions about the health effects of O_3 and CO remain unanswered. I believe it will be important and cost-effective to insure that research on the health effects of these pollutants and their underlying mechanisms be pursued vigorously.

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TESTIMONY
SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION
SENATE COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS
HEARING ON HEALTH EFFECTS OF AIR POLLUTION
APRIL 18, 1989

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Mr. Chairman, I appreciate your invitation to testify on the health effects of ozone (O_3), carbon monoxide (CO), and precursors of acid rain at this hearing. I have been conducting laboratory research on the health effects of acidic aerosols with animals and humans since 1975, and research on the pulmonary effects of ozone in ambient air on humans since 1980. I have also participated in the Clean Air Scientific Advisory Committee (CASAC) reviews of EPA Criteria Documents and Staff Papers for CO, nitrogen dioxide (NO_2), sulfur dioxide (SO_2) and particulate matter, and O_3 since 1980, serving as Chairman of CASAC between 1983 and 1987. During the final stages of our review of the agency's documents on particulate matter in 1985, CASAC requested that the Agency prepare an Issues Paper on the health effects of acidic aerosols. We viewed acidic aerosols, i.e., sulfuric acid and ammonium bisulfate, as the most likely components of particulate matter to be a causally related to the excess mortality and morbidity which have been associated with elevated concentrations of particulate matter. The CASAC Subcommittee on Acidic Aerosols reviewed the Issues Paper in June 1988 and, based upon this review, recommended that the Administrator consider listing acidic aerosol as a new criteria pollutant.

I recently prepared an extensive review of the health effects of the six criteria pollutants for the Congressional Research Service (CRS), and the Committee Staff has made copies of it available to the members of the Subcommittee. This testimony summarizes the health effects of O_3, acidic aerosols, and CO without citations to the supporting literature. The citations are available in the CRS paper. My testimony also includes a summary of some critical factors affecting human exposures to these pollutants and some of the technical factors and policy options that need to be addressed if current exposures and their health effects are to be reduced in a timely and cost-effective manner. My key findings and recommendations are underlined in the text which follows.

In my view, O_3 and acidic aerosols are currently having the greatest impacts on public health of any of the ambient air pollutants. Being secondary pollutants, i.e., pollutants formed within the atmosphere from gaseous precursors, their concentrations are elevated over extremely broad regions of the country, and exposures are often higher, and the daily peaks more prolonged, in regions 25 to 250 miles downwind of major population centers than within then. Within the cities themselves, nitric oxide from motor vehicles and power plants scavenges O_3, and ammonia from biological decay neutralizes acidic aerosols to the

relatively innocuous ammonium sulfate. Thus, urban monitors may underestimate regional average exposures, and control strategies based upon local source inventories may continue to be as unrealistic and ineffective as they have proven to be for O_3 in recent decades.

Health Effects of Ozone

Controlled human inhalation exposure studies in chambers have demonstrated that acute effects of inhaled O_3 include decrements in lung function, increases in respiratory symptoms, increased reactivity to other irritants and antigens, increased membrane permeability, depressed particle clearance from the lungs, and an influx of cells associated with lung inflammation. These studies have been conducted at levels of exposure comparable to those occurring frequently in ambient air. At any given O_3 concentrations, the severity of the responses increases directly with duration of exposure and the volume of air inhaled. Thus, the greatest responses to O_3 occur among people who spend many hours out-of-doors engaged in vigorous work or play. Working groups at risk include construction workers, farm workers, postal workers, police officers, athletes, etc. Other groups at risk include those engaged in strenuous recreation, including adult joggers and tennis players, as well as children of all ages engaged in vigorous play activities.

Most environmental health scientists have relied upon the exposure-response data from controlled exposure tests conducted on volunteers in chambers containing O_3 in otherwise pure air as being indicative of the acute effects of O_3 in ambient air. In one such test performed in the EPA's Clinical Studies Branch, 22 healthy young men were exposed to 0.08 ppm O_3 for 6.6 hrs, including 5 hours of moderate exercise. Respiratory function, as indicated by the forced expiratory volume in one second (FEV_1), decreased with each succeeding hour of exposure, and at the end of the study was reduced by an average of 7%, with one individual having a 26% reduction. Greater responses were produced with O_3 concentrations of 0.12 ppm, as illustrated in Figure 1. EPA staff has proposed, and CASAC has agreed, that the effect becomes adverse when the magnitude of the change exceeds 10% in an individual, and 7 of the 22 individuals exposed at 0.08 ppm crossed that threshold. The 6.6 hour O_3 exposure studies clearly demonstrate that the 1 hour averaging time in the current ambient air quality standard is inappropriate, and should be replaced with a 6 to 8 hour averaging time in future standards.

While such studies conducted in exposure chambers with O_3 in purified air provide the most precise data, but they indicate lesser effects than those which occur in natural settings, where O_3 is a component of a mixture of air pollutants. In a study that I directed, involving 30 young adults who exercised outdoors at Tuxedo, NY for 1/2 hour each day during 4 weeks of the summer of 1985, the FEV_1 decrements associated with ambient O_3 were about 3/4 as large as those seen after 6.6 hours of chamber exposure. Thus, the effects were accumulating at a much greater rate than in the chamber. Similarly, the effects we have seen in our 1980, 1982, 1984, and 1988 studies of children at summer camps have been much greater than those seen in children exposed to O_3 in purified air

in chambers. We suspect that the potentiation of the characteristic O_3 response in these field studies is due to acidic aerosols, but further research is needed before this hypothesis can be confirmed.

The acute effects of O_3 are largely, but not entirely reversible within one day, and the contributions of these acute effects to chronic lung disease are not known. There is good evidence that repetitive O_3 exposures, at levels comparable to those frequently encountered in ambient air, produce chronic lung damage in rats and monkeys. The lungs become less elastic, and lung capacity is reduced. One prospective epidemiological study conducted in two communities in Southern California reported that lung function loss among nonsmokers over five years was greater in the community with the higher O_3 levels. It is also of interest to note that the rate of function loss in the community with the lower O_3 exposure was much greater than that reported by others for communities with O_3 levels that are much lower than those in the low exposure California community. The animal exposure data and the epidemiological data are insufficient at this time to permit any definitive assessment of the extent of chronic effects in humans, but they are at least consistent with each other, and with the evidence that acute exposures produce an inflammatory response. Considering that millions of people are being exposed chronically to high levels of O_3, serious concern about accelerated lung aging is warranted, and additional research focussed on the chronic effects of O_3 is clearly of high priority.

Health Effects of Acidic Aerosols

Acidic aerosols can also have both acute and chronic health effects, but the data bases on both levels of ambient exposure and responses to acute exposures among humans are much less extensive than those for O_3. Much of the exposure-response data base is suggestive, rather than conclusive because surrogate measures of exposure, such as black smoke (used in London), and SO_2 vapor or the sulfate ion content of particulate matter in the U.S., were used rather than direct measurements of aerosol hydrogen ion. In recent years, levels of acidic aerosol, measured as hydrogen ion have been made in the U.S. and Canada, indicating that concentrations in excess of 20-50 $\mu g/m^3$ (as sulfuric acid) have been observed for time durations ranging from 1 to 12 hours. The first data on annual average acidic aerosol concentrations in contemporary U.S. communities were recently reported for four eastern U.S. communities in the Harvard Six-Cities study, with the highest being 1.8 $\mu g/m^3$ (as sulfuric acid).

The human health effects of major concern with respect to the inhalation of acidic aerosols are bronchospasm in asthmatics, and acute and chronic bronchitis in all exposed persons. The former relates to acute exposure, while the latter can be related more closely to chronic or cumulative exposures. In either case, the effects are produced by droplets depositing on the surface of the conductive airways of the lungs.

Studies at the University of Washington related to the provocation of bronchospasm show evidence for increased airway resistance in exercising mild to moderate asthmatics following sulfuric acid exposures

for 30 minutes at rest and 10 minutes of exercise for adolescents at 68 or 100 $\mu g/m^3$. Similar effects were seen in adults exposed at the University of Rochester for 2 hours at 75 $\mu g/m^3$ while exercising, or for 16 minutes at 450 $\mu g/m^3$ while at rest. While the average increases in airway resistance were small, the populations studied were small, and some subjects had much greater responses than the average. Also, the populations were carefully selected and did not include the more unstable and potentially more reactive asthmatics in the population.

Studies of the effects of sulfuric acid aerosols on tracheobronchial and alveolar rates of particle clearance at New York University Medical Center showed that the effects increased with exposure duration and concentration. Sulfuric acid on ultrafine particles which simulate coal combustion effluent produce functional responses about 10 fold greater than those produced by pure droplets of sulfuric acid of comparable size in studies performed at M.I.T. The acute effects are more closely related to cumulative daily exposure than to peak concentrations. Thus, an exposure limit for sulfuric acid aerosol should be expressed as a multihour average rather than as a short-term concentration limit.

With respect to concentration limits to protect against chronic bronchitis, a mechanistic basis for linkage between chronic exposure to sulfuric acid aerosol and the pathogenesis of chronic bronchitis was established in the series of chronic exposure studies at NYU. Persistent alterations in lung structure in rabbits were associated with changes in particle clearance function. These functional changes are, in turn, indicative of increased mucus secretion, leading to mucus stasis, a hallmark of bronchitis disease.

The animal studies can be related to human responses in two ways. One is the concordance in functional and morphometric responses of animals to sulfuric acid and cigarette smoke, a known causal factor for human chronic bronchitis. The other is that humans and animals have essentially the same transient mucociliary clearance function responses to acute exposures. Daily one hour sulfuric acid exposures in animals produce persistent changes in clearance function, and it is highly likely that humans would also if similarly exposed. Furthermore, a comparison of human and rabbit responses to single exposures indicates that humans respond at lower concentrations.

The effects of sulfuric acid aerosols on the airways are very likely to be cumulative within each exposure day. Thus, the daily one-hour exposures at 250 $\mu g/m^3$ in the rabbits may be equivalent to < 50 $\mu g/m^3$ for a seven- to eight-hour day and to a still lower concentration for equivalent effects in humans.

Several recent studies strengthen the basis for associating chronic inhalation exposures of ambient acidic aerosols with bronchitis in humans. The most direct evidence was presented for four U.S. cities in the Harvard Six-Cities study which varied in annual average ambient acid concentrations from 8 to 35 $nmoles/m^3$ (0.4 to 1.8 $\mu g/m^3$ of sulfuric acid equivalent). The incidence of reported symptoms of bronchitis in the

last year among 10-12 year old children varied in proportion to the ambient acid concentration (Figure 2). Indirect, but supportive evidence was developed following an acidic pollution episode in Jan. 1985 in the Ruhr district in West Germany in which average concentrations of SO_2 and suspended particles were 800 and 600 $\mu g/m^3$. There were significant increases in deaths, hospital admissions, outpatient visits, and ambulance deliveries to hospitals in comparison to those in a less polluted control area.

Pulmonary function was measured in primary school children in the Netherlands before, during, and after the Jan. 1985 acidic pollution event which caused elevated exposures throughout northwestern Europe. During this episode, the Dutch children were exposed to respirable particulate and SO_2 concentrations in the range of 200-500 $\mu g/m^3$. Their baseline respiratory functions had been measured about 4-6 weeks earlier, when the pollutants were below 100 $\mu g/m^3$. During the episode, function indices were significantly lower by 3-5 percent, and decrements were still present at 16 days later, but not at 25 days.

Associations between 1980 U.S. mortality rates in 98 Standard Metropolitan Statistical Areas (SMSAs) and four measures of particulate air pollution were reported by investigators at Harvard University. The four index pollutants were total suspended particulate matter (TSP), inhalable particulate matter, i.e., particulate < 15 μm in aerodynamic median diameter (IP), fine particulate matter, i.e., particulate < 2.5 μm in aerodynamic median diameter (FP), and sulfate ion ($SO_4^=$), a major component of FP. Annual mean $SO_4^=$ concentrations in this data set varied from ~ 3 to 20 $\mu g/m^3$. FP and $SO_4^=$ were most consistently and significantly associated with the reported SMSA-specific total annual mortality rates, while TSP and IP were often non-significant predictors of mortality. Figure 3 is my adaptation of the results of the SMSA-mortality analyses, showing a hypothetical extrapolation of the analytical results based upon the hypothesis that the acidic aerosol component of the particulate is the most likely causal factor for the demonstrated associations between human mortality and morbidity and exposure to airborne particulate matter.

Several studies have shown significant statistical associations between British Smoke, an index of black particulate pollution, and daily mortality in metropolitan London during the winters from 1958 to 1972. The associations of mortality with SO_2 in these studies were weaker or absent. In a re-examination of London, England mortality in relation to exposure to acidic aerosols during 1963-1972 winters at NYU, acid aerosol concentrations were more closely correlated with daily mortality than were either British Smoke or SO_2. The results were similar for all years and for each winter separately. A typical winter was 1965-66, with an average acidic aerosol concentration, expressed as sulfuric acid, of 7.5 $\mu g/m^3$, and with a maximum daily value of 24.3 $\mu g/m^3$.

The association between acute bronchitis in children and annual average acid aerosol concentrations averaging < 2 $\mu g/m^3$ (as sulfuric acid) suggests that an ambient air quality limit should be less than 1 $\mu g/m^3$ as an annual average. The association between excess daily

mortality, primarily among elderly persons with chronic obstructive pulmonary disease, and sulfuric acid aerosol concentrations < 25 $\mu g/m^3$ suggests that a maximum 8 or 24 hour ambient air quality limit should be below 10 $\mu g/m^3$.

Health Effects of Carbon Monoxide

In comparison to O_3 and acidic aerosols, the health effects of carbon monoxide (CO) are well known, and there is relatively little controversy about them. As an outdoor pollutant CO is almost entirely attributable to motor vehicle exhaust. Motor vehicle exhaust can also be a dominant source indoors, especially in residential units and public buildings with internal garages. Sidestream cigarette smoke can also contribute significantly to indoor CO concentrations. Unvented indoor combustion sources such as gas ranges and space heaters, can cause very high concentrations of CO when they are not properly maintained or operated. However, they contribute relatively little CO under normal usage.

Outdoor CO concentrations exceeding the current ambient air standard of 9 ppm for 1 hour maximum and 35 ppm for 8 hour maximum occur in the central cores of large metropolitan areas, due to the concentration of motor vehicles in confined street canyons. As a result, exposures of people above the standard can be expected among people who spend a major fraction of their time out-of-doors in central cities during peak traffic hours. Other people likely to have excessive exposure are those who work in or near parking garages, or those who live in urban areas at high altitudes such as Denver and Albuquerque, where the partial pressure of oxygen is reduced and the motor vehicle fuel is burned less completely.

Cardiovascular system effects are attributed to CO. Aggravation of angina pectoris by CO is an adverse health effect for at least two major reasons. First, the angina reflects an oxygen deficit which may also result in cardicvascular damage that is unquantifiable, using present technology. Second, aggravation of angina may be the first in a series of progressively more serious symptoms that accompany cardiovascular disease. CO exposures resulting in carboxyhemoglobin (COHb) levels of 2.7 to 2.9% were linked to adverse health effects in some portion of the estimated five to seven million persons with angina pectoris in the U.S. population, making such persons or "susceptible population", providing the basis for the current ambient standard. Recent clinical studies performed under the Health Effects Institute program have provided further supporting evidence for the validity of this standard.

Population Exposures and Options for Control

While the general perception is that O_3 is present at concentrations of concern only in California, this is not the case. Approximately half of the U.S. population lived in communities with ambient ozone concentrations which exceeded the current standard of 0.12 ppm for 1 hour (equivalent to 0.10 ppm for 8 hours) at least once per year during the period 1983-1985. Figure 4 shows that daily peak exposures are

spread over 8 or more hours. More than 10 million people are exposed to such concentrations while exercising at moderate to heavy levels of exertion and hence subject to at least the transient effects cited above. In 1987 and 1988, O_3 concentrations were higher than those in the 1983-1985 period, and the population exposure was therefore larger. One reason why so many people are exposed to O_3 at levels which elicit functional responses and symptoms is that O_3 continues to form in the atmosphere far downwind of the sources of hydrocarbons and nitrogen oxides. This widespread ozone contamination increases both the number of people exposed and the magnitude of their exposure in warm weather, when the largest number of people are outdoors for extended periods. Similar considerations apply to acidic aerosols.

Our ability to develop effective and efficient control strategies is currently severely constrained by our limited knowledge of the physics, chemistry, and meteorology of O_3 and acidic aerosol formation, transport, and degradation. Two key points that are not fully appreciated are:

1. The background peak concentration of O_3 in remote regions of North America, of about 0.040 ppm, is — 1/3 of the current standard, and is rising — 0.001 ppm per year. This increase may be related to the worldwide increase in atmospheric methane of anthropogenic origin.

2. The absence of sufficient data on the concentrations of nitrogen oxides (NO_x) and hydrocarbons from natural sources, as well as on the emissions and concentrations of anthropogenic hydrocarbons, NO_x, O_3, formaldehyde, hydrogen peroxide and other species at remote sites, severely limits the ability of air quality models to provide a reliable basis for constructing effective control strategies for O_3. Similarly, the factors affecting the oxidation of SO_2 vapor to sulfuric acid aerosol, and those leading to the neutralization of acidic aerosols by ammonia vapor are not yet well established.

In summary, there are widespread areas of the U.S. with sufficient O_3 in ambient air to elicit demonstrated health effects, and that there are very large numbers of people repeatedly exposed to such levels. Due to the narrow range between rising background levels and the effects levels within the current standard, enormous expenses and disruptions will be required to control hydrocarbon and NO_x emissions enough to reduce O_3 exposures to levels below the standard. Furthermore, the recent research findings suggest that: 1) the current standard has the wrong averaging time and has no margin of safety, and therefore is too lenient to satisfy the intent of the current Clean Air Act; and 2) that current control strategies are not likely to be adequate for reducing O_3 to target levels in many U.S. communities for many years. It is therefore important that the public understand the issues affecting the implementation of the Clean Air Act by EPA, and its possible revisions according to proposals being considered in the Congress. In the near term, we must decide whether to enforce the existing Act as it is, to fail to enforce it, or to modify it. Under the existing Act, a new O_3

standard would need to be proposed in the Federal Register in the near future. It is almost certain that the proposed standard would be either the same as the current standard, or more stringent, as recommended by the EPA staff and the Clean Air Scientific Advisory Committee. Full compliance with the current standard, let alone a more stringent one, cannot realistically be expected within this century in all U.S. communities, while attempts to enforce such compliance using current strategies would require major costs and disruptions in many.

The second option, that is to retain the current Act's provisions, but fail to enforce them for O_3, should not seriously be considered. We should not have laws that can be ignored without consequences.

The third option, that is to change the provisions of the current Act, has to be considered carefully, but with the recognition that the other two options are very unattractive. Ozone could be given a special exemption from the provisions of the current Act, since early enforcement of the Act for the other criteria pollutants is feasible. A precedent of sorts is the exemption of saccharin from the provisions of the Delaney amendment to the Food and Drug Act. Under this option, there would be no unnecessary reduction in public health protection for the other air pollutants. The issues to be addressed would then be how to formulate the provisions of a revised Act dealing with the protection of the public health and welfare arising from exposure to O_3. This could include the issuance of advisories and guidelines for individual actions to minimize exposure. EPA has adopted this approach for residential radon exposures.

Timetables and Research Needs

A rational national policy on tropospheric O_3 control must be formulated and implemented on a reasonable time scale, to allow the application of: improved control technologies; alternate transportation options; knowledge of the physics, chemistry, and meteorology of O_3 formation, transport, and decay; and knowledge of the chronic health effects of O_3 exposure. It should encourage industry to develop alternate fuels for internal combustion engines which can be demonstrated to have less pollution impact. As suggested by recent regulatory decisions in Southern California, these might include methanol, compressed natural gas, and hydrogen. It should also encourage the development of compact and efficient electrical energy storage for electrically driven motor vehicles and/or compound systems with continuous steady-state, low emission combustion and onboard storage of surge power in electrical batteries or flywheels. Less polluting transportation options such as those enumerated above should not be rejected just because they might require 10-or-more years to implement. In the interim, the EPA should prepare and disseminate guidance to the public on means of minimizing exposure, such as exercising outdoors before 10 AM when O_3 concentrations are usually minimal.

While long-term pollution reductions options should be encouraged, it is also important to take reasonable and prudent steps in the meantime to reduce precurser emissions from other sources and overall

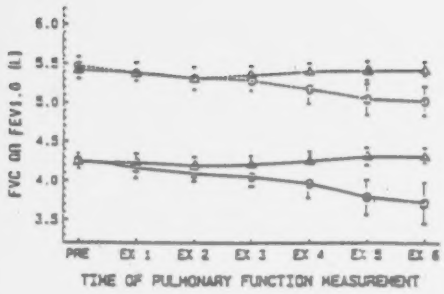
exposures to O_3 and acidic aerosols, and to establish new research programs to generate the data needed to properly guide future decisions that will have to be made in issuing health advisories and choosing control options. Priority areas for further research include: 1) the effects of O_3 and acidic aerosols on the pathogenesis of chronic lung disease; 2) the potentiation of characteristic health effects of O_3 by acidic aerosols and other environmental cofactors; 3) the influence of methane, natural hydrocarbons, and nitrogen oxides on background and peak O_3 concentrations, and the use of such data in improving the reliability and utility of air quality models used for developing control strategies.

During the period when interim steps are being taken to reduce exposures to O_3 and acidic aerosols and their effects, EPA should have the mandate and means to conduct and support the research needed to design and implement the longer term pollution prevention and health protection goals of the Clean Air Act. The critical issues before us, i.e., the formation and transport of the secondary air pollutants, and their chronic health effects, cannot be effectively addressed in highly targeted short-term research. These are complex problems needing a long-term research perspective more typical of the National Institutes of Health than of EPA. Congress needs to recognize that EPA needs the authority and funding to address longer term research goals. It needs to be able to commit to programs that will take up to 5 years or more to complete, and to protect the funds for these programs from diversion to the short-term regulatory agenda which now drives the EPA research program.

Conclusions

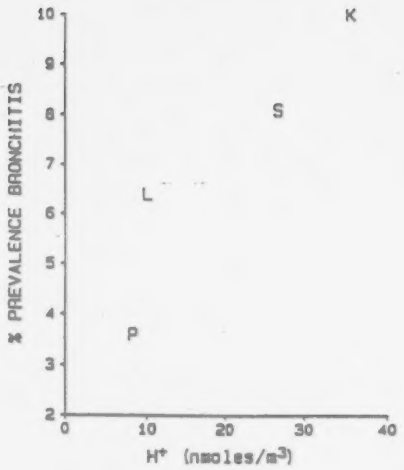
In conclusion, the health effects of O_3 exposures permitted under the current standard produce lung function decrements greater than 10% in some healthy normal humans and are clearly adverse under the current Clean Air Act. An exemption of O_3, allowing it to have a more permissive standard, could have serious health and welfare consequences, such as restrictions on outdoor recreation during the summer, or premature aging of the lungs. Most state and local governments cannot adequately control the hydrocarbon and nitrogen oxide emissions which determine their populations' exposures to O_3, or the emissions of sulfur dioxide which determine exposures to acidic aerosols. Thus, any further shift of responsibility to local government for source control would be ineffective. It could also lead to more interstate disputes and litigation, as has already been seen in the acidic precipitation control controversy. We must recognize that a program to control ambient O_3 and acidic aerosols to levels which protect against adverse health effects will require a long, difficult and expensive national campaign. We need to educate the public about the problem we face in dealing with O_3 and acidic aerosols, and how they can minimize their exposures in the interim. With public support and understanding, this difficult challenge can be met.

FIGURE 1.



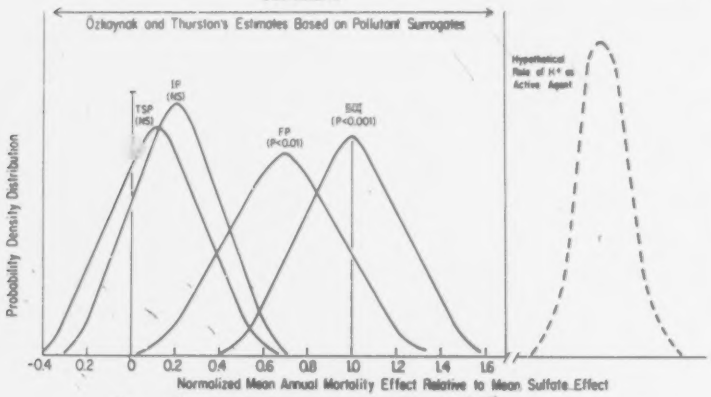
Mean ± S.E. of forced vital capacity and forced expired volume in one second after each of six exercise periods. Dashed lines are FVC and solid lines are FEV 1.0; triangles represent air exposures and circles ozone exposures. From: Folinsbee et al.

FIGURE 2.



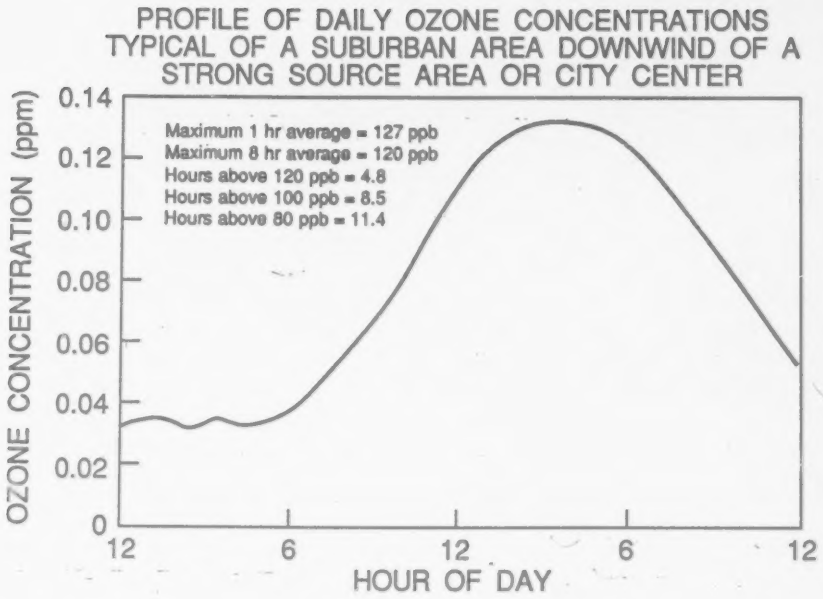
Bronchitis in the last year in children 10 to 12 yrs of age in four U.S. cities by annual average concentration of hydrogen ion. (P = Portage, WI; L = St. Louis, MO; S = Steubenville, OH; K = Kingston, TN). From: Speizer.

FIGURE 3.



Comparative estimates of mean mortality effect of chronic exposure to TSP, IP, FP, and $SO_4^=$ particle concentration measures for 96 SMSA Model of Ozkaynak and Thurston (43), and hypothetical effect of aerosol hydrogen ion concentration. From: Lippmann.

FIGURE 4


PROFILE OF DAILY OZONE CONCENTRATIONS
TYPICAL OF A SUBURBAN AREA DOWNWIND OF A
STRONG SOURCE AREA OR CITY CENTER
OZONE CONCENTRATION (ppm)
0.14
0.12
0.10
0.08
0.06
0.04
0.02
0
Maximum 1 hr average = 127 ppb
Maximum 8 hr average = 120 ppb
Hours above 120 ppb = 4.8
Hours above 100 ppb = 8.5
Hours above 80 ppb = 11.4
12
6
12
6
12
HOUR OF DAY

KEY FINDINGS AND RECOMMENDATIONS
IN TESTIMONY OF
MORTON LIPPMANN, Ph.D., PROFESSOR OF ENVIRONMENTAL MEDICINE
NEW YORK UNIVERSITY MEDICAL CENTER, TUXEDO, NY 10987
AT HEARING ON HEALTH EFFECTS OF AIR POLLUTION - 4/18/89
SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION
SENATE COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS

1. Ozone (O_3) and acidic aerosols are currently having the greatest impacts on public health of any of the ambient air pollutants.

2. The greatest responses to O_3 occur among people who spend many hours out-of-doors engaged in vigorous work or play. Working groups at risk include construction workers, farm workers, postal workers, police officers, athletes, etc. Other groups at risk include those engaged in strenuous recreation, including adult joggers and tennis players, as well as children of all ages engaged in vigorous play activities.

3. The 6.6 hour O_3 exposure studies clearly demonstrate that the 1 hour averaging time in the current ambient air quality standard is inappropriate for protection against acute responses, and should be replaced with a 6 to 8 hour averaging time in future standards.

4. Studies conducted in exposure chambers with O_3 in purified air provide the most precise data on acute effects, but they indicate lesser effects than those which occur in natural settings, where O_3 is a component of a mixture of air pollutants.

5. Repetitive O_3 exposures, at levels comparable to those frequently encountered in ambient air, produce chronic lung damage in rats and monkeys. The lungs become less elastic, and lung capacity is reduced.

6. Considering that millions of people are being exposed chronically to high levels of O_3, serious concern about accelerated lung aging is warranted, and additional research focussed on the chronic effects of O_3 is clearly of high priority.

7. The human health effects of major concern with respect to the inhalation of acidic aerosols are bronchospasm in asthmatics, and acute and chronic bronchitis in all exposed persons.

8. The association between acute bronchitis in children and annual average acid aerosol concentrations averaging $< 2\ \mu g/m^3$ (as sulfuric acid) suggests that an ambient air quality limit should be less than $1\ \mu g/m^3$ as an annual average. The association between excess daily mortality, primarily among elderly persons with chronic obstructive pulmonary disease, and sulfuric acid aerosol concentrations $< 25\ \mu g/m^3$ suggests that a maximum 8 or 24 hour ambient air quality limit should be below $10\ \mu g/m^3$.

9. Priority areas for further research include: 1) the effects of O_3 and acidic aerosols on the pathogenesis of chronic lung disease; 2) the potentiation of characteristic health effects of O_3 by acidic aerosols and other environmental cofactors; 3) the influence of methane, natural hydrocarbons, and nitrogen oxides on background and peak O_3 concentrations, and the use of such data in improving the reliability and utility of air quality models used for developing control strategies.

10. EPA needs the authority and funding to address longer term research goals. It needs to be able to commit to programs that will take up to 5 years or more to complete, and to protect the funds for these programs from diversion to the short-term regulatory agenda which now drives the EPA research program.

11. Most state and local governments cannot adequately control the hydrocarbon and nitrogen oxide emissions which determine their populations' exposures to O_3, or the emissions of sulfur dioxide which determine exposures to acidic aerosols.

12. A program to control ambient O_3 and acidic aerosols to levels which protect against adverse health effects will require a long, difficult and expensive national campaign.

T E S T I M O N Y

BEFORE THE

U.S. SENATE COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS
SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION

ON

THE HEALTH EFFECTS OF AIR POLLUTION IN NON-ATTAINMENT AREAS

PRESENTED BY

BAILUS WALKER, JR., Ph.D., MPH

April 18, 1989

Washington, D.C.

My name is Bailus Walker, Jr., Ph.D., MPH. I am Professor of Environmental Health and Toxicology, Graduate School of Health Sciences, State University of New York, Albany, and Immediate Past President of the American Public Health Association. I was a member of the National Academy of Sciences' Committee on the Epidemiology of Air Pollution. A report of our study was submitted to the Congress and copies are available at the Academy.

I am today representing the American Public Health Association -- the world's oldest and largest organization of health professionals and I am pleased to discuss the health effects of selected air pollutants in non-attainment areas.

No reputable member of the scientific community questions the potential risk to human health of air pollution. For all pollutants, there is a limit beyond which health and life are threatened. This common-sense conclusion is borne out both by accidental exposure of individuals and by certain community exposures.

Acid precipitation -- an old problem whose growth has been insidious -- is now a serious environmental health (broadly defined) problem. It is a phenomenon, not a toxic substance per se, but it can increase the risk of human exposure to air pollutants and toxic metals, including the following:

-- sulfur dioxide -- both as sulfur dioxide gas and the transformation product, sulfate;

-- nitrogen oxides -- as a gas, as the transformation product nitrate, and as a precursor of ozone;

-- reactive hydrocarbons -- as a precursor of ozone.

One measure of the magnitude of the problem is the annual emissions of the major precursors of acid precipitation. During 1987, approximately 26 million tons of sulfur dioxide, 21 million tons of nitrogen oxide, and 24 to 28 million tons of hydrocarbons were emitted from man-made sources. Over 80 percent of these emissions were from sources in 31 states bordering or east of the Mississippi River.

If we add to this load the estimated 4.8 billion pounds of toxics -- including 235 million pounds of carcinogens such as benzene and formaldehyde, and 527 million pounds of such neurotoxins as toluene and tricholorethylene (TCE) -- annually released into the air, it is clear we are confronted with a

major environmental health problem.

Almost 102 million people in the United States reside in counties which exceed one or more air quality standards. Several counties in the far west, the midwest and the east still exceed ambient standards for sulfur dioxide (SO^2). Here we know from a large volume of health-effect studies that excessive levels of sulfur dioxide in the ambient air are associated with acute and chronic respiratory disease.

Sulfur dioxide is a respiratory irritant that is absorbed in the nose and upper respiratory tract. Public health studies have found an increase in lung cancer mortality in men and increases in bronchitis in men and women who were exposed long term to sulfur dioxide. Sulfur dioxide aggravates existing conditions such as asthma, and heart diseases. During even brief exposures to SO^2, asthmatic individuals experience impeded movement of air in and out of the lungs. At very low concentrations, they may experience difficulty in breathing; "wheeze most days and nights." Moreover, exercise potentiates the effects. Once in the atmosphere, SO^2 is converted to sulfate particles. Two main types are known to be produced: sulfuric acid and ammonium sulfate. Both types of particles are extremely small and may be inhaled deep into the lungs.

Sulfuric acid aerosols have a variety of adverse health effects: spasm of the larynx (the organ of voice) and of the lungs which may cause death, depending on concentration and individual sensitivity.

Populations at special risk from particulate sulfates are the elderly and adults with pre-existing chronic heart or lung disease. Children are especially susceptible to increased lower respiratory tract illness and to decreased lung function.

Many analyses of cities with different air pollution levels comparing death rates among specific populations have shown a strong correlation between pollution levels and death rates. Evidence is strongest for correlation between acute episodic effects and severe air pollution incidence. The combination of sulfate particles and other associated particles in the atmosphere may cause severe health damage and death. Indeed, this exposure may cause approximately 50,000 premature deaths per year in the United States and Canada.

Nitrogen oxides -- the term refers to a number of compounds: nitrogen oxide (NO), and nitrogen dioxide (NO^2), and such secondary by-products as nitric acid and nitrate aerosols.

Los Angeles County is the only county that has not attained the air quality standard for nitrogen oxide -- a deep lung irritant capable of producing severe lung damage.

Infants and children exposed to atmospheric conditions in Los Angeles County experience a higher than "normal" rate of acute respiratory infections.

Unfortunately, not enough is known yet about the effects of nitrogen oxides (NO_x) to permit a precise quantitative estimation of the health-related damages for which NO_x may be responsible.

There are some indirect health effects of acid precipitation which must not be overlooked. Acid precipitation may increase human exposure to several potentially toxic metals by increasing metal concentrations in major pathways, particularly food and water and, in some cases, the conversion of metal species to toxic forms.

Acidified waters can dissolve such toxic metals as aluminum, copper, lead and mercury. It can also release such toxic substances as asbestos from soil and rocks in watersheds, lakes and from drinking water distribution systems.

Of particular concern is lead and mercury. Acidification of drinking water sources significantly increases lead content. Leaded water pipes become particularly hazardous as water pH (acid) decreases. And the effects of lead on brain function are increasingly reported. These effects include adverse effects on children's behavior and intelligence. These data prompted the Centers for Disease Control to lower the definition of an elevated blood lead level in children from 30 to 25 ug/dl.

Mercury can occur as several species. Of concern here is methyl-mercury which has a tendency to bioaccumulate in nature -- a tendency dependent on acidification. The effects of methyl-mercury on the fetus are the best documented example of a metal compound that affects the development process of the central nervous system including postnatal growth retardation.

As with many other aspects of the health effects of acid deposition, further studies are needed to more precisely identify human exposure to metal toxicity provoked by acid deposition.

In summary, air pollution is one of the greatest risks to public health in the United States. It causes, contributes to, or aggravates a long list of disease and dysfunction -- chronic bronchitis, lung cancer, nervous disorders and heart disease. As many as 50,000 premature deaths may be caused by single air pollutants or a combination of pollutants.

Indeed, by EPA's own calculations (based on 20 chemicals), air toxics (benzene, etc.) alone cause more than 2,000 cases of cancer every year.

Acid deposition in water sources can dissolve metals and toxic substances from soil, rock and pipes and transport them to drinking water and into the food supply.

While the most recent air quality emissions trends report published by the U.S. Environmental Protection Agency indicates progress over the years, we must, as a nation, do far more to reduce the risk of air pollution-provoked disease and dysfunction.

The American Public Health Association strongly urges Congress to (1) strengthen the Clean Air Act with the necessary amendments which you will be considering; (2) allocate the resources needed by the regulatory agencies -- federal and state -- for strict enforcement of the Amended Act; and (3) maintain careful oversight to ensure that the goals of the legislation are met.

The atmosphere constitutes our most intimate environmental contact. As such, it must promote human health and not destroy it.

Thank you.

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AMERICAN LUNG ASSOCIATION
The Christmas Seal People ®

STATEMENT OF

THOMAS GODAR, M.D.

ON BEHALF OF

THE AMERICAN LUNG ASSOCIATION

TO THE

SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION

SENATE COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS

RE: HEALTH EFFECTS OF AIR POLLUTION

APRIL 18, 1989

Mr. Chairman, members of the Subcommittee, I am Dr. Thomas Godar, Director of the Pulmonary Disease Section at St. Francis Hospital and Medical Center, Hartford, Connecticut. I am also Associate Professor of Medicine, University of Connecticut School of Medicine and Yale University School of Medicine and in the active practice of chest medicine. Today I am presenting comments as the President of the American Lung Association. Based on our knowledge of the adverse health effects of air pollution, the American Lung Association places a very high priority on preserving and strengthening the federal government's air pollution control program.

Although other witnesses have been asked to review the specific health impacts of exposure to the various criteria air pollutants, I would like to take a moment to briefly comment on the striking advances made in information concerning the health effects in humans from experimental exposures to ozone. When the American Lung Association first appeared before this Subcommittee, appended to our comments was a "state of the art" review entitled, Health Effects of Air Pollution, published by the American Lung Association's medical section, the American Thoracic Society. An updated version of that report is nearly complete and includes the following recent key findings:

o Health Effects of Exposure to Ozone Containing Atmospheres:

  o Ozone can cause immediate, short term changes in lung function and increased respiratory symptoms in healthy adults and children who exercise moderately or heavily during periods of elevated ozone concentrations. Significant decreases in lung function

NOTE: Attachments to this statement have been retained in committee files.

and symptoms such as coughing and pain upon deep inspiration are experienced by exercising individuals exposed to ozone for one to two hours at levels comparable to those frequently found in many nonattainment areas.

- Lung function decrements have been found in particularly sensitive exercising people exposed to ozone levels at or just below the current one hour standard of 0.12 parts per million.

- Significant changes in lung function and increased frequency of respiratory symptoms have also been observed at concentrations significantly lower than the current one hour standard of 0.12 parts per million when exposures last for longer periods of time -- approximately seven hours.

In addition to these acute, short term effects, there is growing medical concern that repeated exposures to ozone may result in structural damage to the lungs at or levels below the current standard. Such exposures are suspected in the development of chronic lung diseases. Although some studies suggest that persistent health effects are caused by repeated exposure to ozone, our knowledge in this area remains limited and more study is imperative.

- Health Effects of Exposure to Sulfur Dioxide Containing Atmospheres
  - SO_2, at levels as low as .25 ppm for five minutes can cause

the development of significant increases in airways resistance -- bronchoconstriction -- in asthmatics under conditions involving moderate levels of exercise.

- sulfuric acid aerosols also provoke bronchoconstriction in exercising asthmatics exposed to concentrations which can be found in ambient air pollution.

- Health Effects of Exposure to Nitrogen Dioxide Containing Atmospheres

  - NO_2, at concentrations near levels of current peak ambient air pollution, cause effects on lung function of asthmatics.

  - NO_2 exposure has been linked with increased frequency and severity of respiratory tract infections in animal studies and epidemiologic surveys.

- Health Effects of Exposure to Airborne Particulate Matter

  - Acid aerosol particulates are a highly likely causal factor for effects on lung structure and function and have been associated with morbidity and premature mortality.

Populations at Risk

The protracted air pollution problem is of considerable concern to the

American Lung Association. Approximately 62 percent of all Americans --150 million people -- breathe air containing pollutants that exceed one or more of the health-based National Ambient Air Quality Standards.

The Clean Air Act requires that the National Ambient Air Quality Standards be set with "an adequate margin of safety" to account for gaps in scientific information on the health effects of criteria pollutants, with particular concern for those population sub-groups that are potentially at higher risk from exposure to these pollutants. The American Lung Association quantified these sub-groups and has included this data in a report entitled, Breath in Danger.

ALA's report provides an estimation of the populations at risk of adverse health consequences in all areas not in attainment with the National Ambient Air Quality Standards. The report is appended to my comments and I would like to request that it be included in the hearing record. ALA used as its definition of populations at risk the following definition as described by EPA:

". . . Populations at risk is a segment of a defined population exhibiting characteristics associated with significantly higher probability of developing a condition, illness, or other abnormal status. This high risk may result from either greater inherent susceptibility or from exposure to situations peculiar to that group. What is meant by inherent susceptibility is a host characteristic or status that predisposes the host to a greater risk of heightened response to an external stimulus or agent."

Estimates of the populations at risk in areas not in attainment for carbon monoxide and ozone were derived from EPA data assembled as a necessary step in complying with the Mitchell-Conte amendment contained in the Budget Reconciliation Act of 1987.

For ozone, the ALA report includes in the populations at risk the following:

- persons with pre-existing respiratory disease, including asthma and Chronic Obstructive Pulmonary Disease. This group is at particular risk because they already have limited lung function and may not be able to tolerate even small decrements in lung function that are otherwise tolerable to the healthy person;

- elderly persons, 65 years of age and older. This group is at particular risk because their relatively deficient immune system caused by aging magnifies the increased susceptibility to respiratory infections resulting from exposure. An area needing further study is effects in the healthy elderly exercising population;

- pre-adolescent children, 13 years of age and younger. This group is at particular risk because their lung defense mechanisms are not yet fully developed because their breathing rate and therefore the dose of pollutants to which they are exposed is far greater than that of adults, and because any early damage to still-maturing lungs may be magnified in later life and become the cause of chronic lung

disease.

Our estimates for ozone are conservative since we have not included in the analysis the sub-group of people referred to as "responders"--the 5 to 20 percent of exercising, healthy, normal adults and children that have an increased response to ozone in comparison to the general population.

For carbon monoxide, the ALA report includes in the populations at risk the following groups:

- pregnant women, whose unborn children are potentially at risk. The fetus is at particular risk from carbon monoxide because its oxygen supply is low and dependent on exchange with the mother's blood. Carbon monoxide exposure threatens the tenuous chain which supports the fetus;

- persons with pre-existing coronary heart disease. This group is at particular risk because their heart --already weaken by disease -- is particularly sensitive to the displacement of oxygen from the blood stream by carbon monoxide resulting in pain upon mild exertion.

Our analysis for counties classified as not in attainment for ozone and carbon monoxide estimates the magnitude of the at-risk populations as follows:

- There are 354 counties that are either classified as not in attainment for ozone or proposed for such classification, with an estimated total

of 123,071,100 persons potentially exposed to dangerous levels of ozone; sub-groups particularly at risk in these areas are:

- o 25,040,599 pre-adolescent children;
- o 15,370,604 elderly;
- o individuals with asthma including 1,289,837 pediatric asthmatics and 3,582,742 adult asthmatics; and
- o 6,074,222 people with Chronic Obstructive Pulmonary Disease.

- o There are 214 counties that are either classified as not in attainment for carbon monoxide or proposed for such classification, with an estimated total of 89,028,300 persons potentially exposed to dangerous levels of carbon monoxide; sub-groups particularly at risk in these areas are:

  - o 1,870,659 coronary heart disease patients; and
  - o 1,410,053 pregnant women.

These estimates emphasize the need for timely and effective legislative and regulatory action to address the pervasive nature of the current air pollution problem. Such action is especially important in protecting the health and well-being of the significant number of individuals who are particularly at risk of adverse health consequences and can least afford exposure to airborne pollutants.

The recent EPA air quality monitoring data for 1988 add further to the concern

the American Lung Association has for the quality of our nation's air resources. ALA reviewed this data and developed estimates of the most vulnerable of the individuals living in these 28 new areas with ozone levels above the health-based standard. The population of these communities include the following sub-groups at particular risk of adverse health consequences:

- o 2,926,242 pre-adolescent children;
- o 1,788,258 elderly;
- o individuals with asthma including 158,874 pediatric asthmatics and 733,216 adult asthmatics; and
- o 916,298 people with Chronic Obstructive Pulmonary Disease.

These are gross estimates based on the total population in these 28 areas. City-specific data has been added to our report, Breath in Danger.

The Health Costs of Air Pollution

The American Lung Association published The Health Costs of Air Pollution in August, 1985. This report reviewed studies which examined the economic costs related to health problems and premature death from air pollution. A generalization drawn from the results of the studies reviewed in this survey is that total health care costs in the United States from exposure to criteria pollutants are likely to fall in the range of the low tens of billions of dollars. The studies reviewed place a national health cost bill from exposure to the two or three most widely investigated pollutants between $5 billion and $40 billion. Key findings include:

- a 60 percent reduction in average 1978 particulate and sulfur dioxide emissions in the United States would save between $5.1 billion and $15.9 billion in annual health costs related to premature deaths. Reduced sickness or morbidity costs would be worth about $25 billion a year;

- The 20 percent reduction in average particulate and sulfur dioxide air pollution in the U.S. between 1970 and 1978 saves about $13.9 billion a year in mortality-related costs and about $2 billion a year in morbidity costs;

- Reductions in particulate and sulfate air pollution achieved between 1970 and 1978 solely because of pollution control requirements set by the Clean Air Act save about $4.4 billion a year in mortality-related health costs;

- Implementation of a program designed to reduce sulfur dioxide emissions in 11 European countries to levels 34 percent of those in 1974 would increase the average life expectancies of individual national populations by as much as 562 days. Health cost savings related to reduced morbidity alone would be reduced by $5.8 billion a year through this program;

- A program to reduce particulate and sulfur dioxide emissions at two power plants in the Tampa Bay region of Florida would produce morbidity health cost benefits of $5.1 million a year

compared with an annual cost of the program of $3.1 million. If the pollution abatement program were extended to a third power plant in the region, however, total morbidity cost benefits of $5.4 million a year would fall below the annual cost of the program estimated at $9.3 million;

- Programs to reduce 1977 average particulate and sulfate air pollution in six urban California regions would save 850 people each year from premature deaths resulting from exposure to air pollution. Mortality health cost benefits from those programs would be $400 million a year;

- Reducing the average nationwide concentration of sulfur dioxide by one microgram per cubic meter would result in a reduction in morbidity health costs of $20 million a year;

- Reducing the average nationwide concentration of particulate or sulfur dioxide air pollution by one microgram per cubic meter would produce mortality health cost benefits worth between $10 and $25 per person per year. A similar reduction in sulfate pollution would benefit each American between $75 and $150 per year.

Why NOx?

Air quality in America has improved since the Clean Air Act was passed in

1970. Most cities enjoy visibly cleaner air today as a result of the Act. However, the success to date has fallen far short of the national goal of healthful air quality for all Americans -- most of urban America and many other American communities continue to experience pollution levels -- summertime smog -- above the health-based standards. Data on pollution trends suggest that the improvements of the past have hit a plateau or worse, have begun to reverse themselves. The 1988 data suggest a problem that can no longer be ignored.

Earlier this month, Mr. Chairman, the American Lung Association provided you with a briefing on our latest report, Why NOx? The report reviews the public health and environmental indictment against oxides of nitrogen and assesses traditional and emerging control technologies. The conclusions and recommendations are appended to my testimony.

Scientists are finding new health and ecological damage from NOX, confirming long-held suspicions about NOX as a major air pollution problem. Yet, NOx remains the least controlled of the so-called criteria pollutants. NOx is an essential ingredient -- often the essential ingredient -- of virtually every major air pollution problem including ozone or summertime photochemical smog, acidic particulate matter, acid rain, and global warming of the atmosphere. In fact, many believe that NOx control is the key to clean air -- the missing link of the clean air puzzle.

Overall NOx emissions in the United States have remained relatively stable between 19 million and 21 million tons per year for the past decade despite

controls on NOx emissions from cars. NOx emissions from power plants have increased 42 percent in the past 10 years and are expected to double again in the next 20 to 30 years. Total NOx emissions are expected to climb by 50 percent in this period.

NOx control technology is already available and in wide use in Japan, Germany, The Netherlands, and the State of California.

- California is implementing a program to cut NOx emissions from new cars by an additional 60 percent at a cost of approximately $40 per vehicle.

- Selective catalytic reduction technology for NOx from power plants would reduce emissions by up to 90 percent. Use of this technology could result in a 4 million ton reduction in NOx over the next 10 years at an annual cost of approximately $3 per person.

Reauthorization of the Clean Air Act

Congressional action is long overdue on revisions and improvements to the Clean Air Act. To achieve healthful air quality, Congress must provide strong direction to federal, state, and local decision makers. Legislation to reauthorize the Act must include:

- new emission standards for autos and trucks which take maximum

advantage of the technology improvements of the 1980's;

- o new deadlines for nonattainment areas to achieve the health-based standards, with deadlines set to assure that each area takes concerted action to achieve healthful air quality as soon as possible;

- o requirements that state implementation plans for ozone nonattainment include NOx controls;

- o requirements for "90 percent controls" for NOx on motor vehicles and power plants;
  and

- o programs to attack ozone nonattainment on a regional basis, especially for the severely polluted northeast region of the country.

* * * *

The American Lung Association is committed to achieving reauthorization of a strengthened Clean Air Act. To this end, we continue to offer our scientific and medical expertise to assist you in assessing these complex health issues and the appropriate changes needed in the Act for their resolution.

American Academy of Pediatrics

T E S T I M O N Y

BEFORE THE

SENATE COMMITTEE ON ENVIRONMENT & PUBLIC WORKS

SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION

ON

THE HEALTH EFFECT OF AIR POLLUTION IN NON-ATTAINMENT AREAS

PRESENTED BY

PHILIP J. LANDRIGAN, M.D., M.Sc., F.A.A.P.

April 18, 1989

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My name is Philip J. Landrigan, M.D., M.Sc. I am Professor of Community Medicine and Director of the Division of Environmental and Occupational Medicine at the Mount Sinai School of Medicine in New York City. I am a pediatrician, and a member of the Committee on Environmental Hazards of the American Academy of Pediatrics. In 1985, I was Co-Vice Chairman of the Committee on Epidemiology of Air Pollutants sponsored by the National Research Council of the National Academy of Sciences.

I am representing the American Academy of Pediatrics, and I am pleased to discuss the effects of air pollution on the health of children. The Academy believes that air pollution causes serious and widespread health problems. It is estimated the more than 28,000,000 preadolescent children reside in communities where attainment with one or more national ambient air quality standards of the Clean Air Act is not achieved. I will focus on the health hazards of several air pollutants, including ozone, carbon monoxide and precursors of acid rain.

Ambient concentrations of many air pollutants, such as sulphur dioxide and particulates have decreased sharply over most of the United States since passage of the Clean Air Act in 1970. However, standards for only six hazardous air pollutants have been adopted out of a priority list of 37 hazardous air pollutants scheduled for possible regulatory action. High local concentrations of air pollutants are seen in certain cities in relation to busy highways and industrial complexes. Sulfate concentrations, although lower than in the past, are still unacceptably high in many areas, particularly during the summer months. Finally, and perhaps most important to this morning's hearing, levels of two air pollutants, nitrogen oxides and ozone have not declined. Ozone concentrations have in fact shown some recent increases.

The preponderance of evidence continues to suggest that exposure to air pollution in concentrations frequently found in urban areas, causes illness and premature death. To the extent that health effects of different pollutants are additive, the actual health effects of all pollutants to which people are exposed could be much higher than those suggested by limited studies. A survey conducted by the American Lung Association of studies published from 1978-1983 estimated that the total health costs in the United States from exposure to key air pollutants now regulated by the Clean Air Act are likely to fall in the range of the low tens of billions of dollars.

I can assure you that ozone, sulfates and the oxides of nitrogen cause disease in children and aggravate preexisting respiratory conditions. This is quite significant considering the prevalence of pediatric pulmonary diseases.

In 1987, 767,000 children under 15 years of age were discharged from a hospital following treatment of a disease of the respiratory system. The length of hospital stay for pulmonary disease is longer than the average for all hospitalizations of children. In the 1985 National Ambulatory Medical Care Survey, the second largest proportion of office visits for the total population surveyed was for respiratory disease. Asthma and chronic bronchitis are two of the more chronic pulmonary diseases in children. Incidence of asthma in children has risen 25 percent from 1982-1986. The rate for hospital discharges with asthma as a primary diagnosis also increased about 50 percent from 1979-1986. Annual deaths from asthma have also risen. Asthma attacks can be triggered by a variety of factors, environmental pollutants among them.

One of the most noticeable and immediate consequences of the drought of 1988 was a dramatic degradation of air quality, particularly in the eastern United States. High temperatures and stagnant air resulted in the formation of record levels of ozone in many urban and rural areas. EPA data from 1988 indicated that there were 26 new urban and 2 new rural areas in which the National Ambient Air Quality Standard for ozone was exceeded for the first time.

Ozone is a highly reactive, oxidizing gas that is formed principally by the action of sunlight on mixtures of nitrogen oxides and volatile organic chemicals released into the air by both stationary and mobile sources. Passage of the Clean Air Act gave legislative authority to the Environmental Protection Agency to set standards for air quality, including ozone and carbon monoxide. Despite the existence of these standards, air pollutants and ozone in many areas of the country are still found at levels that adversely affect public health.

Ozone, a respiratory irritant, causes inflammation and irritability of the airways, coughing, tightness of the chest, pain on inspiration and difficulty breathing. Inhalation of ozone can cause changes in lung function and probably provokes asthma attacks in some individuals. The ability to perform sustained exercise is adversely affected by exposure to ozone. Other symptoms reported less consistently with ozone exposure include headache and nausea.

Nitrogen dioxide is a yellowish brown gas which can be fatal at high concentrations. At lower concentrations NO2 is a lung irritant that can contribute to the development of bronchitis and pneumonia. NO2 can lower resistance to influenza and other lung diseases by interfering with the body's immune system.

Controlled studies of human volunteers in environmental exposure chambers indicate that some people are much more likely to react to ozone than others: this susceptibility

appears to be a reproducible, intrinsic, individual characteristic. Children are more vulnerable than adults to pollutants of any kind. The cellular immaturity of children and their ongoing growth processes account for this elevated risk.

Children's airways are much more narrow than those of an adult's. Thus, minor irritation caused by air pollution, which would produce only a slight response in an adult, can result in a dangerous level of swelling in the lining of the narrow airway of a child. Moreover children's airways are small and children have markedly increased needs for oxygen for their size. They breathe more rapidly and inhale more pollutant per pound of body weight than do adults. If during the early years of life a child is highly exposed to air pollutants, the risk of long-term damage to the lungs increases.

Another factor which makes children more vulnerable than adults to air pollutants is their increased frequency of respiratory infections. The interactions of the infectious process with irritating pollutants intensify the damage to the airways. Furthermore, the processes for repair of damaged airways are less efficient in children than in adults because of the small caliber of the airways and the resultant partial obstruction to the flow of air.

Extensive research has been undertaken to determine the level of exposure to ozone at which adverse respiratory effects occur. These studies have examined clinical symptoms and subclinical changes caused by ozone. Such subclinical changes might not be observed upon physical examination, but can be detected with pulmonary function studies. Changes in lung function and respiratory symptoms have been reported in people exercising for 1 or 2 hours in controlled exposure studies using ozone concentrations as low as the federal hourly standard of 0.12 ppm. Exercise or other vigorous activity increases the dose of ozone reaching the lungs, resulting in the occurrence of symptoms and changes in pulmonary function that might not occur in people at rest. Recent research reported by EPA staff indicates that longer exposures can cause respiratory effects at ozone concentrations as low as 0.08 ppm.

This recent work supports previous field studies of children in summer camps, in which multi-hour outdoor exposures to ozone at concentrations even below 0.12 ppm resulted in transient changes in lung function. A report published in January, 1989 from the Harvard School of Public Health showed an association between ambient levels of ozone less than 0.08 ppm and decreases in children's lung function. In another study, exposure to a four-day exceedance of the ozone standard resulted in pulmonary function changes in children that persisted for up to a week after the episode ended. The delay in full recovery suggests the presence of inflammation

and microscopic airway damage. Such damage persists for hours or days after exposure to ozone has ended and may initiate the development of chronic pulmonary injury and chronic lung disease.

The Harvard-Six-City study has found that exposure to acid air pollution, particularly to fine particulates associated with total sulfate particulates, produces respiratory symptoms in school children. A marked rise in prevalence of bronchitis occurs as the concentration of fine airborne particles increases. This effect is seen both in children with a prior history of wheezing, as well as in children without such history. However, the effect is greater among children who wheeze.

In a series of studies conducted in Southwestern Ontario between 1974 and 1978, associations have been sought between airborne concentrations of particulates, sulfates, oxides of nitrogen and ozone and the frequency of hospital admission in children. In the months of July and August, excess admissions to hospitals for respiratory conditions were significantly correlated with levels of sulfate and ozone, as well as with temperature. Additionally, admissions due to asthma were found to be strongly correlated with ozone levels. No significant relationship was found between air pollution and admission to hospital for any cause except respiratory illness.

The warm, sunny days of summer that favor ozone formation also encourage long periods of outdoor play and sports among children. Children's high levels of activity put them at risk for acute respiratory effects due to ozone exposure. Furthermore, the long-term consequences of repeated exposure to ozone are unknown, but potentially worrisome. Animal studies indicate that chronic exposures can result in persistent low-grade inflammation in the terminal airways, similar to that seen in the lungs of young cigarette smokers. Although the structural changes in the lungs of experimental animal are not as severe as those seen in long-term smoking, the inflammatory response suggests that repeated ozone exposure may result in chronic disease or deterioration of lung function. A recent study of Los Angeles residents suggests that prolonged residence in areas with high ozone concentrations is associated with an accelerated decline of lung function, which could be described as premature aging of the lung.

There is also the question of the extent to which ozone interacts with other airborne agents. There is a substantial body of experimental evidence in animals that ozone can lower resistance to infection, facilitate sensitization to airborne allergens, and act synergistically with airborne acidity in causing damage to the deep lung. These issues have not or cannot be studied in humans, but there are published data that ozone and sulfur-containing particles may interact in

exacerbating respiratory symptoms, as evidenced by increased hospital admissions for respiratory diseases on summer days when ozone and sulfate concentrations are elevated.

The epidemiologic data indicate quite clearly that current atmospheric levels of ozone, sulfate, and particulates are capable of producing transient decrements in pulmonary function of children. These decrements in function may last longer than the episodes of pollution themselves and can persist for at least a week after cessation of the pollution episodes. These decrements in function appear to be associated with increased frequency of asthma and, at least during the summer months, with increased frequencies of hospital admission.

In conclusion, all of these data confirm that exposure to relatively low levels of air pollution at the current federal standard can produce toxic, pathologic effects in the lung, respiratory symptoms and changes in pulmonary function. Experimental studies and some epidemiologic evidence suggest that repeated exposures to ozone may result in chronic lung disease. Ozone may exacerbate the harmful effects on the lungs of infections and other allergenic and toxic airborne agents. These changes occur in people engaged in vigorous aerobic activity, comparable in intensity to popular sports such as soccer, basketball, swimming, jogging and cycling. Acute changes in pulmonary function have been shown to occur in children exposed to ozone at concentrations below the hourly standard. In view of their activity patterns, children are one of the groups in our society most likely to have significant, prolonged ozone exposures.

The current strategies in the United States for attaining clean air are not working. The continued failure to achieve the federal ambient air quality standard for ozone has a negative effect on the health of our nation's children. The task confronting the regulatory agencies today is to exercise their mandate to protect the public health. To delay action is poor medicine and poor public policy.

The American Academy of Pediatrics urges Congress to demand that strict measures be implemented to decrease concentrations of harmful air pollutants. Furthermore, these measures must be vigorously enforced. There are few elements more basic to the health of our children that ensuring that the air they breathe is clean.

Thank you. I shall be pleased to answer questions.

Record Statement of

General Motors Corporation

to the

Subcommittee on Environmental Protection

of the

Senate Committee on Environment and Public Works

Presented by

Dr. Christopher C. Green

April 18, 1989

Washington, D. C.

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Mr. Chairman, I am Christopher C. Green, Head of the Biomedical Science Department at the General Motors Research Laboratories in Michigan. Before coming to GM, I was a Medical Officer in the Federal Senior Executive Service. I am also licensed to practice medicine in the State of New Mexico and the District of Columbia. I am accompanied by Dr. Elaine S. Wright, who heads our pulmonary toxicity research programs.

Mr. Chairman, General Motors recognizes the Clean Air Act's statutory obligation that all areas of the country comply with the Act's health-based standards for ozone and carbon monoxide. We also recognize that, according to EPA's most recent, although preliminary, data for 1988, a number of areas around the country continue to be classified as nonattainment.

Mr. Chairman, I am not here this morning to discuss regulatory programs aimed at achieving attainment. Rather, I am here at your invitation, as both a physician with clinical training and a physiologist with research background to present to the Subcommittee General Motors' views on the potential health effects of ozone and carbon monoxide, two of our highest priority research programs. I am hopeful I can make a contribution to the Subcommittee's understanding of the medical data surrounding this important issue, and the nature and the severity of the health effects associated with the current nonattainment situation.

NOTE: Attachments to this statement have been retained in committee files.

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Ozone

With regard to ozone health effects, I'd like to begin by making three observations. First, we applaud EPA research programs under way in an effort to gain a better understanding of the ozone health effects issue. We have similar kinds of research ongoing in our own laboratories and we have enjoyed scientist-to-scientist opportunities to work with the Agency.

Second, in examining existing data on potential ozone health effects, it is important to note that responses measured in high-dose acute animal laboratory studies at concentrations greater than .7 ppm should not be used to predict effects of ambient concentration, to which the general population is exposed.

Third, care must be taken in interpreting measurable responses at low level exposures, so that we do not place inordinate weight on measurable changes that are within the range of a given subject's individual responses but which lack clinical significance. In our view, it is inappropriate to base health effects judgments solely on measurable changes absent clinical significance.

In defining what constitutes "adverse respiratory health effects," we support the statement of the American Thoracic Society, which defines adverse respiratory health effects in epidemiologic studies as (1) interference with normal activity, (2) episodic respiratory illness, (3) incapacitating respiratory illness, or (4) permanent or progressive respiratory injury or

dysfunction. (See Attachment I: Am. Rev. Respir. Dis. 1986; 131: 666-668.) We also support the EPA's definition of adverse respiratory health effects, as submitted in its OAQPS draft Staff Paper, which recommends that mild ozone responses not be considered an adverse respiratory health effect. (See Attachment II.)

Relating this definition to our laboratory research and our review of existing evidence, including our review of EPA's criteria document on ozone and never data from our laboratory and many others, we do not believe that clinically significant health effects have been shown to result solely from exposures to ozone concentrations at levels represented by EPA's most recent nonattainment data.

Small deviations in the measurement of lung function of individuals during unusually heavy exercise have been produced with exposures to ozone concentrations at lov levels. The reasons for these responses are, however, unclear. For instance, EPA's criteria document points out, there is a groving body of experimental evidence that suggests that such responses represent the involvement of sensory receptor responsiveness, especially of the upper airvay, the nose, throat, and in the bronchial tree. That is to say, such responses may simply be a transient reaction to ozone exposure by the body's nervous system. If this is correct, it is difficult to accept that such signs and symptoms constitute an adverse health effect, as opposed to a normal homeostatic, or protective, physiological response.

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A critical factor in ozone responsiveness is a person's level of exercise at the time of exposure. As physical exertion draws more air into the deep lung, it draws more ozone. In studies performed at the EPA Clinical Research Branch facilities in Chapel Hill, North Carolina, young healthy male volunteers were exposed to the current standard of .12 ppm ozone for 6.6 hours in environmentally controlled chambers with heavy exercise. These subjects exercised at a level which far exceeds what most people could sustain for even a few hours. While the data indicate variations in lung function that are statistically significant, the studies also showed that such variations are fully reversible. It is important to note that other similar studies demonstrate that such changes in lung function do not occur without heavy exercise.

The Clean Air Act contemplates that sensitive subpopulations at greater risk must be identified and protected. In this regard, research efforts, our own included, have focused on individuals having pre-existing disease, such as those persons who suffer from asthma. We agree with the EPA criteria document's findings that, based upon currently available evidence, individuals with pre-existing disease respond to ozone in about the same way as do normal, healthy individuals. Indeed, patients with chronic obstructive lung disease and/or asthma have not shown increased reaction to ozone in controlled human exposures even at low ambient levels of ozone.

Additionally, recent studies have failed to support the assertion that the elderly are at greater risk from ozone exposure. In controlled chamber studies, Drechsler-Parks, et al, used standard pulmonary function tests to

compare the responses of elderly men and women (aged 51 to 70 years) to those of younger men and women (aged 18 to 30 years). (See Attachment III.) Subjects were exposed to a fairly high ozone concentration, .45 ppm, for two hours, with intermittent moderate exercise. In these experiments older men and women were far less responsive to ozone, than the younger subjects. Other studies confirm these findings generally, and there are no published studies which contradict them.

In similar studies undertaken in our Biomedical Research Department, normal healthy, young men and otherwise healthy asthmatics of the same age were exposed to clean air and also to .40 ppm ozone for 2 hours with periods of moderate to heavy exercise. The asthmatic subjects did not use medication for 36 hours prior to the test in order to maximize the possibility of detecting an effect. The normal subjects as a group showed no response to clean air, while asthmatics responded mildly to exercise even in the clean air chamber. Lung volume changes were the same for both groups. Asthmatics, however, showed greater airway obstruction than normals in both clean air and in ozone. (See Attachment IV: Kreit, J. W., Gross, B., Moore, T. B., Lorenzen, T. J., D'Arcy, J., and Eschenbacher, W. L. "Ozone-induced changes in pulmonary function and bronchial responsiveness in asthmatics". Journal of Applied Physiology 66, 217-222; 1989.) Other studies at lower ozone concentrations and using asthmatics who were not deprived of their medication, have not shown this differential response.

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As we review the literature we find that other studies have shown that, 1) like the elderly, cigarette smokers and individuals with chronic obstructive pulmonary disease, appear to be less sensitive to ozone effects than normal individuals; 2) Children have not shown greater effects than normal, healthy adults in either clinical or epidemiology studies; 3) Epidemiologic studies performed to date have not identified an excess sensitivity to ozone among asthmatics and only one controlled exposure study has demonstrated differences in response of asthmatics to ozone compared to normal adults. In short, there is little evidence, in spite of a great deal of study, to suggest that age or pre-existing respiratory disease predisposes individuals to excess susceptibility to ozone effects in today's ambient environment.

With regard to purported adverse health effects in children exposed to ozone, the significance of several summer camp studies need to be addressed. First, the epidemiological observations in these studies have not always produced consistent results. Moreover, the alleged trends in pulmonary function tests were not significant enough to document ozone-induced injury or an adverse health effect in the respiratory system. Citing statistical differences in such subtle changes of physiological functions without noting the clinical significance, can create a false impression regarding the importance of the findings. For example, comparable responses and symptoms are produced by many other factors, such as high heat and humidity, in our day-to-day activities. These, like the symptoms from low level ambient exposures, rapidly disappear when the exposure is discontinued.

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Finally, with regard to concerns about potential long-term effects, existing evidence does not support claims that responses observed in animals exposed to ozone concentrations above .5 ppm, are representative of the kinds of effects that might be found if the animals were exposed to the lower ambient concentrations at which human exposure occurs. In fact, the most recent evidence shows that contrary to previous predictions, long-term exposures of laboratory animals to low concentrations of ambient ozone do not result in any significant fibrotic changes in the lung. (See Attachment V: Wright, E. S., Kehrer, J. P., White, D. M. and Smiler, K. L., " Effects of Chronic Exposure to Ozone on Collagen Metabolism in Rat Lungs" (1988). Toxicology and Applied Pharmacology: 92, 445-452.) These studies used low concentrations of .12 ppm ozone and reported that contrary to predictions that had been extrapolated from much shorter experiments with higher concentrations, the animals tolerated practically a lifetime of exposure without any significant harmful effects on lung collagen content or synthesis; that is, the protein material believed to relate to the fibrosis mentioned above.

Other long-term studies conducted in our laboratories have also shown that unless the day-to-day exposures to ozone are higher than .5 ppm, inhalation produces minimal changes in lung function and even these minimal changes rapidly disappear when the exposure ceases. (See Attachments VI and VII: Kenneth B. Gross and Harold J. White, "Pulmonary Functional Morphological Changes Induced by a Four-Week Exposure to .7 ppm Ozone Followed by a Nine-Week Recovery Period," J. Tox. and Environ. Health 17: 143-157, 1986; and "Functional and Pathological Consequences of a 52-week Exposure to .5 ppm Ozone Followed by a Clean Air Recovery Period," Lung. 165, 283-295; 1987.)

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These experiments raise serious questions regarding interpretations of other animal studies as being indicative of permanent lung injury due to lov concentrations of ozone.

Carbon Monoxide

Regulation of CO has been affected by a number of controversial scientific studies. The controversies over health effects of exposure to various levels of carbon monoxide first involved data from the laboratories of Beard and Wertheim which claimed to identify central nervous system effects after inhalation of lov levels of CO. This study has long been discredited. The controversy then turned to data from the laboratories of Aronov and covorkers which purported to relate CO levels to aggravation of angina. These studies claimed that small amounts of carboxyhemoglobin (COHb) in the blood of angina patients causes a decease in their ability to withstand extended exercise without experiencing angina discomfort. The most controversial claim by Dr. Aronov was that levels of COHb as lov as 2% can cause an average of 10% reduction in exercise tolerance in angina patients. Hovever, these studies were likevise questioned.

Subsequently, the Health Effects Institute (HEI), a research institute jointly funded by the EPA and the automotive industry, attempted to reproduce the Aronov study using brief exposures to high levels of CO (350 ppm). The findings of these studies have not yet appeared in the peer-revieved scientific literature. Therefore, the status of the findings of the studies are uncertain at this time. Hovever, careful reviev of the exposure protocols, and the

clinical significance of the observed effects will occur as a natural part of the scientific process. For example, hov the CO levels used in the study relate to real world exposure situations remains to be analyzed. Scientists will evaluate the findings and form opinions about the results. Therefore, the impact of the study on CO rulemaking is uncertain at this time. However, ve look forward to the HEI final report and the possibility that it will enhance the scientific basis for CO regulation.

Our Research Laboratories are involved in several projects on the health effects of exposure to lov concentrations of carbon monoxide. For example, our Biomedical Science Department plans to study the kinetics and mechanisms of CO effects using nevly developed technology. In addition, a cooperative study involving our Biomedical Science Department and our Environmental Science Department will measure the accumulation of carbon monoxide in the blood of Denver motorists in heavy traffic, while their local carbon monoxide environment is being continuously monitored. Another study with Texas Health Sciences Center in the summer of 1989 will document the increase in COHb level in human volunteer subjects which accompanies going from lov to high elevations. The results of these studies will help clarify the important issues in CO toxicity, especially whether or not CO dissolved in the blood rapidly, such as in the early research protocols and others with hundreds ppm, may be causing the effects, rather than in experiences at ambient levels of less than 10 ppm, in which such effects are not seen.

Summary

The scientific community as well as General Motors shares the Subcommittee's interest and concern with regard to protection of public health and nonattainment of the National Ambient Air Quality Standards for ozone and carbon monoxide. However, we believe that the consequences of our nation's failure to meet the NAAQS for these pollutants, with respect to health effects, have been the subject of considerable overstatement. The levels of ozone and carbon monoxide concentrations in nonattainment areas rarely, if ever, reach levels that are known to cause adverse health effects, versus adaptive physiological effects...which themselves revert to normal at the end of exposure.

We recognize the statutory obligation to meet the health-based standards as expeditiously as practicable. We recognize that the data upon which the scientific community relies could be subject to differing interpretations. In many respects it is difficult not to be overly conservative in our efforts to protect public health. The EPA has a regulatory and criteria review process that is currently active in assessing health effects of exposure to various levels of ozone and carbon monoxide. We congratulate EPA in its research efforts to date and look forward to working with the Agency in its review of these most important issues, in a new partnership in which governmental, industrial, and academic medical research is scrutinized through the health scientific process of peer-review. The past year has shown an excellent beginning, which for our part, we pledge to continue with more published research.

Thank you.

Robert F. Phalen, Ph.D.
Director: Air Pollution Health Effects Laboratory
Professor of Community & Environmental Medicine
College of Medicine
University of California, Irvine

The Health Effects of Ozone

My testimony primarily deals with the issue of human health effects associated with ozone exposure at air concentration levels that presently are found in U.S. cities. I am a research toxicologist with a specialty in injury to the lung, and I am the director of the Air Pollution Health Effects Laboratory in the Department of Community and Environmental Medicine in the College of Medicine at the University of California at Irvine. In addition, I have served on several scientific panels that evaluated scientific data on air pollutants for the U.S. Environmental Protection Agency and for the National Institutes of Health. Additionally, I served for 8 years as chairman of the University of California, Irvine, Medical Center's ethics committee for human subjects in research. In that capacity I have reviewed about 2,000 protocols involving human research, so I am very familiar with the applicability of basic animal toxicology studies to the protection of human health.

My personal research involves (a) computer modeling to assess risks to children from airborne substances, and (b) animal toxicology studies focused on ozone and mixtures of ozone with other common air pollutants such as formaldehyde, diesel soot, sulfur dioxide, airborne acids, etc. I would like to discuss four topics from the point of view of my training and research experience concerning the health effects of inhaled air pollutants. It is useful to bear in mind that the average adult breathes 10-20 thousand liters of air per day, but only 1-2 liters of food and about the same amount of water are consumed during the same period. Thus the potential for significant contact with toxic agents by way of the inhalation route is quite large.

Ozone Toxicity: The first topic relates to the direct toxicity of ozone. It is a strong oxidant gas that penetrates deeply to the very delicate tissues (alveoli, or air sacs) of the lung upon inhalation. In numerous studies involving laboratory animal species that have been demonstrated to be good models for human lung tissues the effects observed at our ambient air concentrations are cause for concern. These effects include: 1) the killing of critical cells in the lung, 2) interference with the ability of the lung's self-cleaning mechanisms, 3) a decrease in the lung's ability to kill viruses and bacteria, 4) impairment of the immune system and possible increased expression of lung tumors in susceptible strains of animals, and 5) a loss of integrity of tissues in relation to their leakiness regarding body fluids and foreign chemicals. Several other effects have also been documented in the laboratory in studies that include laboratory animals and

human volunteers. Thus, the scientific evidence is very clear; ozone is harmful to health, even at relatively low concentrations. It is an unusually toxic chemical.

Safety Factors: The second topic involves the need for an adequate safety factor for human populations exposed to ozone. In this country we have an enviable record, compared to many other countries, in protecting the public health from the effects of overexposure to harmful chemicals in our drinking water, food, medications and in the environment. This good record is not due to making assumtions that thresholds will occur at low doses, or to ignoring important toxicological data. Our protection of public health has been due to extensive laboratory toxicity testing, the application of adequate safety factors when translating laboratory animal data into permissible human exposures, and to our long-term follow-up monitoring of human populations that have been exposed to the chemicals in question.

The accepted, and proven, safety factors in this process are seldom less than a factor of 10, and in some cases are greater than 1,000. This means, for example, that large human populations would be permitted exposure to only 1/10 of the smallest dose of a chemical that produced an adverse effect in any laboratory animal species. As was discussed in my 1984 book Inhalation Studies (pg. 214-220), the use of a safety factor of 10 (for medicines) can still be associated with adverse effects in 3% of the exposed human population. This is due to the great variability among humans in body size, metabolism, nutrition, health status and other factors.

Clearly we cannot maintain a safety factor of even 10 for environmental ozone because exposures of rats to concentrations of only 0.2 or 0.3 ppm lead to serious adverse effects. However, we should use as great a safety factor as is possible from the point of view of protecting human health. This translates into the need for rigorous defense of our current ambient air standards, and if possible, even reducing the concentrations now observed in many cities.

Children: The third topic involves young children, a population whose inhalation exposure to air pollutants has been one of my research specialties over the past few years. Several factors are important to consider regarding children. First, they are born with immature lungs and they have inadequate defenses against many environmental pollutants. For example, the typical 6-month old child has about 10 respiratory tract infections per year, and the average between birth and 5 years of age is about 6 respiratory tract infections per year. Adults have only about 2-3 respiratory tract infections annually. Second, smaller people must breathe large amounts of air per pound of body weight in order to maintain their normal metabolism. A newborn at rest, for example, breathes more than 3 times the amount of air per pound than does an adult. This greatly increases the newborn's dose from air pollutants. Another factor is that childrens' air passages are smaller, and

thus collect a larger fraction of the airborne pollutants than those of adults. Small children's doses from airborne contaminants are higher than those of adults--in some cases by 5-10 times or more. The bottom line is simply that children must be considered as an unusually sensitive group in comparison to adults when air pollutants are considered. Adding all of these factors together gives a needed additional safety factor of about 5-10 for small children over that adequate for adults.

Mixtures of Pollutants: The fourth topic relates to mixtures of pollutants. We never breathe air pollutants singly; a mixture is always present in the environment. Most toxicology studies of ozone have involved exposures to ozone alone. The thousands of potential ozone-containing combinations--with acids, soot, oxides of sulfur, oxides of nitrogen, etc.--have only recently begun to be thoroughly examined. In our laboratory we have found that other pollutants do not always intensify the harmful effects of ozone. However, nitrogen dioxide gas does interact strongly with ozone to produce a mixture that is significantly more harmful than either gas alone. I believe that the enhancement of injury is due to the formation of a third pollutant--nitric acid. There is also strong evidence that sulfuric acid intensifies the effects of ozone on the lungs. We must assume that other, as yet untested, combinations with ozone may produce such synergistic actions.

Conclusions: There is no doubt that human populations in the United States are experiencing adverse health effects from the existing air pollutants. Ozone is certainly one of the more toxic of these air pollutants. The results of reliable animal studies clearly show that the types of effects that ozone produces are very deleterious to health. Any erosion of our current air quality standards for ozone would be very unwise from the point of view of protecting public health.

I thank you for the opportunity to present this testimony.

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**STATEMENT OF E. REGIS McFADDEN, JR., M.D.,
TO THE SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION,
SENATE COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS**

For Presentation at Hearing on April 18, 1989

Introduction

In considering the potential health effects of pollutants involved in the "acid rain" phenomenon, particularly sulfur dioxide (SO_2), one of the key issues is whether exposure to these pollutants poses a danger to the health of sensitive individuals such as asthmatics. I have had long experience in treating and studying patients with asthma. I am currently the Argyl J. Beams Professor of Medicine at Case Western Reserve University in Cleveland and Director of the Airway Disease Center at the University Hospitals of Cleveland. My curriculum vitae is attached to this statement. I am familiar with the literature and have published numerous papers on the effects of various substances and stimuli on asthmatics, and have made a particularly careful review of the human clinical studies on the responses of asthmatics to SO_2 exposure. In addition, we have done testing in our laboratory on the effects of various substances including SO_2 on asthmatics.

I appreciate the invitation of this Subcommittee to present my views on the effects of SO_2 on asthmatics. I am making this statement in my capacity as a consultant to the American Mining Congress and the National Coal Association, but

the views expressed in this statement are solely my own, based on my experience in studying and treating asthmatics and on my review of the pertinent studies.

I would like to begin by stating that I am vitally concerned about atmospheric pollution and am anxious to protect my family and patients from any potential harm from this source. Hence, I fully support the need for environmental regulation and fully share the concerns of this Subcommittee on that subject. At the same time, I believe that environmental laws and regulations should be based on good science and that additional air quality controls or standards entailing great costs should not be imposed where, on the basis of the scientific data, they do not appear to be justified to protect the public health or welfare.

Evidence on the Effects of SO_2 on Asthmatics

The principal concern about the effects of SO_2 on asthmatics is that asthmatics will suffer adverse effects from brief exposure to SO_2 at the peak concentrations that occur sporadically around power plants and similar sources. This concern is not based on any epidemiological evidence showing such effects, but rather on a series of laboratory studies, published since about 1980, reporting bronchoconstrictor (i.e., airway constricting) responses in asthmatics exposed to SO_2 for brief periods.

At the SO_2 concentrations involved in these studies -- typically around 1.0 ppm and below -- effects have not been observed in normal individuals, but only in asthmatics. Even in asthmatics, SO_2 alone at these concentrations has not produced any consequences. Rather, in order to produce responses, the exposure must be accompanied by a fairly significant increase in the level of ventilation, usually through moderate to heavy exercise or, in some cases, through voluntary hyperventilation by mouth. Under such conditions, various investigators have reported that the SO_2-exposed asthmatics experienced changes in certain measures of pulmonary function, such as specific airway resistance, and/or symptomatic reactions such as wheezing, shortness of breath or chest tightness (e.g., Bethel et al, 1983a, 1983b; Sheppard et al, 1983; Linn et al, 1983a, 1984, 1987; Schachter et al, 1984; Roger et al, 1985; Horstman et al, 1986, 1988; Balmes et al, 1987).

These responses are reversible and short-lived. They abate rapidly after the cessation of exercise, even when the SO_2 exposure continues (e.g., Schachter et al, 1984; Hackney et al, 1984). The studies show a wide range of responsiveness to SO_2 among asthmatics, with some subjects having larger responses than the mean and others not reacting at all (e.g., Horstman et al, 1986). In general, however, the responses are modest and similar to the types of reactions frequently experienced by asthmatics in response to many common stimuli. Unlike certain occupational

exposures, antigens (pollens, molds, environmental dusts, etc.) or viral infections, SO_2 exposure does not lead to acute asthma attacks that require emergency therapy or hospitalization for relief. Moreover, again in contrast to antigens, SO_2 does not appear to alter the sensitivity of asthmatics' airways to non-specific stimuli in a way that makes their airways more sensitive to external or internal stimuli or that induces episodes of bronchoconstriction over prolonged periods. Nor does it appear to lead to any other cumulative or long-term effects. The latter facts are demonstrated by studies finding no greater response to SO_2 when subjects were reexposed on succeeding days (Sheppard et al, 1983; Linn et al, 1984), those finding no effect of prior SO_2 exposure on bronchial reactivity to cold air (Linn et al, 1987), and those finding no residual effect of SO_2 exposure in the day or week after the exposure (Linn et al, 1983a, 1987; Hackney et al, 1984). Further, a diminished response has been found when subjects were reexposed to SO_2 or reexercised within a short time on the same day (Sheppard et al, 1983; Roger et al, 1985; Kehrl et al, 1987).

The studies also demonstrate that medications which are already widely used by asthmatics for exercise and other stimuli, such as the $beta_2$-agonists and cromolyn, will also protect against bronchoconstriction due to SO_2 (Hackney et al, 1988; Koenig et al, 1987, 1988; Myers et al, 1986). Indeed, the $beta_2$-agonists, which are the recognized therapy for acute episodes of

asthma on a world-wide basis, will not only prevent SO_2-induced bronchoconstriction if taken before the exercise, but also will reverse any such bronchoconstriction that may develop after exercise.

Evaluation of the Significance of the Reported Responses to SO_2

Questions can be raised about the methods and statistics used in many of the studies on the effects of SO_2 on asthmatics. For example, in the study finding the most extreme reactions (Balmes et al, 1987), the subjects were given the SO_2 through a mouthpiece with their noses clipped, while breathing at a high rate. That mode of delivery has been shown to produce greater effects than those that would occur during natural breathing (Linn et al, 1983b; Bethel et al, 1983a). Rather than dwelling on such problems, however, I think it would be more useful to focus on the overall significance of the reported effects.

The pulmonary function changes typically observed in the studies are on the order of a 100-200% increase in specific airway resistance or a 15-20% reduction in FEV_1. Such changes do not show large effects. They are generally within the range of diurnal variations in lung function so common in many asthmatics and are comparable to or less than the changes that could occur in asthmatics on other occasions due to such non-specific stimuli as walking briskly for a bus on a cold day, laughing vigorously,

sighing, running up a flight of stairs, exposure to perfumes or powdered laundry detergents, or eating at a salad bar. It has been my experience from examining and studying thousands of patients that asthmatics typically will not begin to sense bronchoconstriction until their FEV_1 falls about 30% and specific airway resistance increases about 400%. By way of comparison, asthmatic patients coming to a physician or emergency room for treatment of acute exacerbations of their illness often have decreases in FEV_1 of 65-70% from normal and increases in specific airway resistance of 900-1000%.

The symptoms reported are likewise short-lived and similar to the kinds of reactions that asthmatics experience frequently and tolerate well. The subjects' complaints disappear promptly when they are removed from the stimulus, and they do so without any medication or, in a few cases, upon use of a routine inhaled bronchodilator. Use of such a bronchodilator is itself an ordinary part of the asthmatics' experience and cannot be equated with the kind of emergency therapy or medical treatment that would indicate a significant health effect. As previously mentioned, moreover, the exposures do not appear to lead to any exacerbation of the asthmatics' underlying disease activity or other long-term effects. I would thus regard the symptoms as more in the nature of temporary annoyances rather than adverse health effects.

It should also be emphasized that, in order for most asthmatics to engage in serious exercise without getting exercise-induced bronchoconstriction, they must either pretreat themselves with a medication, such as a $beta_2$-agonist, or else treat themselves afterwards with such a medication. In general, an asthmatic with even modest airway reactivity simply cannot avoid airway obstruction from developing at moderate to high levels of ventilation without use of such medication. Since SO_2 produces responses in asthmatics only in conjunction with moderate to high exercise, use of the medication, either before or after the exercise, would necessarily also prevent or ameliorate an effect of any SO_2 that the asthmatic should encounter during exercise.

It has been argued that individuals with more severe asthma than the mild asthmatics generally studied might be more sensitive to SO_2 or suffer greater effects from an SO_2 exposure. There is no evidence to bear out that suggestion. Indeed, it has been found that, while more severe asthmatics are more responsive to exercise alone than are mild asthmatics, they are not more responsive to SO_2 (Linn et al, 1987). Further, the more severe the asthma, the more important it is that the asthmatic use a medication before or after exercise, regardless of the presence of SO_2 in the air, and that medication would also be effective against any SO_2 effect. Asthmatics with a very severe degree of chronic airway obstruction would simply be unable physically to

achieve the relatively high levels of ventilation that appear to be necessary to produce a response to SO_2. As with patients with chronic obstructive pulmonary disease (Linn et al, 1985), those inherent exercise limitations would appear to protect such severely impaired asthmatics from any SO_2-induced effects.

In any event, the data indicate that, in order for an asthmatic to experience a bronchoconstrictive response to SO_2, he or she must be exercising outdoors at a moderate or higher level, must be off medication, must be sensitive to SO_2, and must be exposed to a peak SO_2 concentration of around 0.4-0.5 ppm or higher for a period of at least 2-5 minutes. EPA has found that, with current air quality conditions, such exposures are rare (Env. Prot. Agency, 1988). According to EPA, only about 1% of asthmatics in the country would experience such a peak SO_2 exposure during exercise in a year, and only a further fraction of those exposed asthmatics would experience a response.

By contrast, as noted previously, asthmatics frequently experience similar or greater responses due to a wide variety of factors unrelated to air pollution. In this context, the occurrence of one or two additional responses due to peak SO_2 exposure in a relatively few asthmatics, given the transient nature of such responses, would not make any significant difference to the asthmatics in terms of their activity patterns or health status. It is thus not surprising that there is no epidemiological or other real-world evidence of a relationship

between SO_2 at current ambient levels and adverse health consequences in asthmatics in the general population. Indeed, one epidemiological study specifically looked for an association between peak hourly SO_2 levels and emergency room visits for asthma in New York City, and found no such association (Goldstein and Weinstein, 1986). By the same token, I do not see how the further regulation or control of SO_2 peaks would result in any detectable reduction in the overall incidence of bronchoconstriction in asthmatics or improvement in the public health.

Conclusion

In these circumstances, it is my opinion, on the basis of the available data, that possible brief exposures to SO_2 at the levels in the ambient air do not represent a threat to the public health, including the health of asthmatics. I do not believe, therefore, that costly new air quality controls could justifiably be based on those data.

April 12, 1989

E.R. McFadden, Jr., M.D.

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HUMAN HEALTH EFFECTS OF AIR POLLUTION WITH SPECIAL REFERENCE TO SULFUR OXIDES:

STATEMENT TO THE SUBCOMMITTEE ON ENVIRONMENTAL PROTECTION OF THE SENATE COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS

DONALD F. PROCTOR, M.D.

I have accepted the invitation to present to the Subcommittee on Environmental Protection my views on the human health effects of sulfur oxide air pollution, especially sulfur dioxide. I will do so because of my long time interest in the problem. This statement is submitted in my role as a consultant to the American Mining Congress and the National Coal Association, but it represents exclusively my own views and conclusions on the subject.

As a physician I have been a member of the clinical staff of the Johns Hopkins Medical Institutions for over fifty years. In that capacity and as a physiologist and a teacher of medical students I have become increasingly involved in preventive medicine; and, during the past 35 years I have devoted more and more of my time to the investigation of human health effects of the atmospheric environment and human defense systems against such hazards (Brain, Proctor, and Reid 1977; Proctor and Andersen 1982). A copy of my curriculum vitae is attached as Appendix A.

Through collaboration with Dr. Ib Andersen (now Director of the National Institute of Occupational Health in Denmark) I have concentrated upon the indoor air and on sulfur dioxide. My clinical and investigative activities have of

necessity required a broad interest in air hygiene especially in relation to the route of access of ambient air into the lungs, the nose and throat. My specialization in Otolaryngology and Bronchoscopy has facilitated that interest.

A major part of our research in the School of Hygiene at Hopkins has been directed at defining the fate and effect of inhaled materials. We have concentrated upon upper airway and mucociliary physiology in that connection.

Since the early 1960s there have been many changes in the attitude of scientists and the lay public toward the cleanliness or pollution of the air we breathe. Increasing industrial growth, especially involving the proliferation of a myriad of new chemicals, urban crowding, and, after the energy crisis, the attempts to insulate our living and working quarters from the outdoor air have all contributed to the complexity of the problems confronting us today. In spite of the surge in interest and activity in the attack on those problems it seems to me that the goal of assuring totally "clean" air is not immediately within our reach. If that is so then one task which we must face is to choose which airborne materials offer the greatest threat to human health and address our major efforts toward their limitation or elimination.

Even in this relatively rich and enlightened country there are limitations on the number of appropriately trained research personnel, the number of properly equipped laboratories,

and, especially today in the face of our financial deficit crunch, there are limits to the proportion of our total economy which can be applied to even so desperately important a problem as the health hazards of the air which we and our children are breathing right now. In light of these considerations I put it to you that, in designing legislation regarding the emission of any individual or combination of pollutants, we should compare relative health effects. No one would deny the difference in importance between the well known serious health effects of cigarette smoking and the questionable and relatively mild effect of short term peak ambient levels of sulfur dioxide on a small number of asthmatics who may be exercising outdoors when such a level occurs. I believe the present Clean Air Act specifically directs us to pay special attention to even a small group of those particularly prone to a health hazard, but is it not now evident that, for the present, we cannot afford to spend our by no means inexhaustible resources on minor health problems at the expense of relative neglect of those which are admittedly of more far reaching and more serious impact to health? Let us pursue the goal of truly "clean" air while at the same time clearly defining our priorities.

I have been personally involved in three investigations of the human health effects of sulfur dioxide (Andersen et al. 1974; Andersen et al. 1977; and Andersen et al. 1981). Two of these studies were conducted in the environmental chamber at the

University of Aarhus in Denmark. In one of these studies I was in the chamber with a concentration of 25 ppm SO_2, which is many times higher than any current ambient levels. That exposure was extremely unpleasant to me, although our young healthy volunteers made very little complaint. We have not studied asthmatics; but an eminent physician and physiologist who is an asthmatic spent some time in our chamber during the 25 ppm exposure and developed a moderate asthmatic attack. He has recently visited us in Baltimore and I asked him about this episode. He told me that the attack was mild and had completely cleared within a half hour (that after exposure to 25 ppm SO_2).

In relation to the general public, even including the elderly and those suffering from chronic obstructive respiratory disease (Linn et al. 1985; Reichel et al. 1978), current ambient levels of sulfur dioxide offer no threat to the public health. It is only in connection with a special sub-group of asthmatics that there remains any controversy; and, in view of the intense study in a number of laboratories, the fact that disagreement persists is in itself perhaps an indication that any effects on even this small, special group may not merit immediate action.

Evidence Available Today on Sulfur Dioxide

On one hand we have the indisputable fact that some asthmatics will develop moderate bronchoconstriction during exercise on exposure in a laboratory to levels of SO_2 (around 0.4 to 1.0 ppm) which produce no measurable effects in other

asthmatics or in normal subjects. In evaluating the significance of studies of this problem we must keep in mind that many of the earlier exposures (and some of those more recent) have been through the use of a mouthpiece and noseclip or a facemask. (Sheppard et al. 1980, 1981, 1984; Bethel et al. 1983a, 1984; Balmes et al. 1987; Koenig et al. 1985, 1989). Findings from those papers cannot be reasonably applied to estimates of real life health effects. One group of investigators have stated this problem quite clearly:

> "Several previous studies have examined the pulmonary effects of SO_2 on moderately and heavily exercising asthmatics who inhaled the SO_2 via a mouthpiece or facemask. Mouthpiece breathing is clearly different from natural breathing and produces greater pulmonary effects. Facemask breathing may alter the pattern of breathing and the shape and resistance of the oral and nasal airways and therefore may also not be equivalent to natural unencumbered breathing. Accordingly, studies of unencumbered subjects such as ours and others are necessary for assessing the effects of SO_2 under natural conditions." (Roger et al. 1985).

You may wish to see for yourself by simply pinching your nostrils tightly together and breathing for several minutes through your mouth how artificial such breathing is. I, and others, have written extensively on this subject (Niinimaa et al. 1981; Proctor and Andersen 1982; Proctor 1983; Proctor 1988). People do resort to oronasal breathing during some heavy exercise and during conversation and singing, but inspired air passes nearly equally through the nose and mouth in those conditions. Still there are a number of reports now available showing that some exercising asthmatics do indeed exhibit a degree of broncho-

constriction when exposed to these levels of SO_2 breathing naturally in an environmental chamber.

On the other hand there are important reservations which must be carefully weighed before we decide that the findings in some exercising asthmatics in an experimental laboratory surrounding indicate a public health hazard or justify special consideration in the control of SO_2 emissions.

1. When bronchoconstriction develops in asthmatics indulging in moderate to heavy exercise during SO_2 exposure, the effect is generally quite moderate and usually comparable to or less than the reactions which they are accustomed to experiencing on other occasions on exposure to allergens, stress or anxiety, cold air, exercise itself, or various other stimuli.
2. The symptoms are readily allayed by the use of bronchodilator medication (which they commonly employ under circumstances wholly unrelated to SO_2) prior to or after exercise, temporary cessation of exercise, or simply going indoors. Indoor levels of SO_2 are generally only about half those found outdoors (Kirsch 1978). Several recent studies have demonstrated that clinically reasonable doses of common asthma medications, such as cromolyn, albuterol and metaproterenol, will greatly inhibit or completely

block SO_2-induced bronchoconstriction (Koenig et al. 1987, 1988; Hackney et al. 1988).

3. In any event the symptoms are relatively mild, are short lived, and do not appear to be related to any cumulative or long term adverse health effect. In one paper, Linn et al. (1987) state: "No statistically significant effect of SO_2 on symptoms was found 1 day or 1 week after exposure. Minimal/mild asthmatics showed, on the average, slight responses at 0.0 ppm (attributable to exercise) and increasing responses at increasing SO_2 concentrations. Moderate/severe asthmatics reacted more at 0.0 ppm, but their increments in response with increasing SO_2 concentration were roughly similar to those of minimal/mild asthmatics. Thus, responses to SO_2 per se were not strongly dependent on clinical severity of asthma, nor on SO_2 exposure history during previous weeks."

4. In real life very small numbers of asthmatics are likely to be exposed to the specific set of circumstances in which SO_2 may play a part in their asthma symptoms. A recent study sponsored by EPRI (1988) indicates that asthmatics spend an average of about 2% of their waking hours exercising strenuously outdoors and about 7% exercising moderately. It also

reports that most asthmatics use medication to some degree to relieve symptoms brought on by exercise, stress, passive smoking, etc. In these circumstances, and given the relative infrequency of SO_2 peaks in excess of 0.4 ppm, it would be an unusual event for an exercising, unmedicated asthmatic to encounter such a peak SO_2 level. On the other hand, the EPRI study reports that asthmatics are likely to experience respiratory symptoms unrelated to SO_2 in about 26% of their waking hours.

One of the questions before this Subcommittee is whether the laboratory studies which have shown a measurable decrease in pulmonary function in exercising asthmatics, especially in young adolescents, on exposure to SO_2 demonstrate a need for further controls on SO_2 emissions. In at least one study (Goldstein and Weinstein 1986) an attempt was made to uncover evidence in a free-living population that short term SO_2 peaks were associated with an increased incidence of asthma. They concluded that no such association was found. As a worst case example of this problem we would have to assume that an asthmatic chooses to exercise outdoors without medication and, in doing so, passes through a region where there is a short-term peak in SO_2. The chances of this happening would appear to be small; but even if it occurred and even if the asthmatic experienced a response, the unhappy individual would merely need

to stop exercising for the moment, use the bronchodilator medication which he employs frequently under circumstances where SO_2 plays no part, or simply go indoors. To my knowledge, there is no epidemiological or other documented evidence of adverse health effects actually occurring in asthmatics in the real world on account of short term exposure to SO_2.

An interesting statement appears in one of the most authoritative recent texts in a chapter written by Woolcock and Permutt (1986):

> "Inhalation of cigarette smoke, inert particles, and sulfur dioxide causes changes in lung function tests in normal humans. These changes, indicating increased bronchial smooth muscle tension, are usually small, occur rapidly, and are rapidly reversed."

I have recently discussed this matter with Dr. Permutt, who expressed the opinion that money would be better spent in supporting basic research on asthma than in further reducing the current levels of SO_2.

Altogether I am forced to conclude that the laboratory studies finding a relationship in some circumstances between SO_2 exposure and modest bronchoconstriction in asthmatics are of academic interest but that those findings have little bearing on the public health. I prepared a report on this subject for EPA in 1985. My review of work done since that report leaves me with the conviction still that there is no scientific justification from the point of view of the public respiratory health for further controls on SO_2 emissions.

Acid Aerosols

Concern has also been raised about the potential health effects of the particulate transformation products of SO_2, especially sulfuric acid aerosols and other acid sulfate aerosols. I claim no particular expertise on the subject of acid aerosols but I do have a long standing interest in the subject of deposition of particles in the respiratory tract (Brain and Valberg 1979; Brain, Proctor and Reid 1977; Proctor and Andersen 1982). Conclusions regarding particulate air pollution including acid aerosols and human health must remain tentative at present. On one hand it is difficult to define particulates in terms of their sources and chemical and physical composition and, on the other hand, our knowledge of their likelihood of deposition within human airways is still a matter for investigation. These problems are particularly difficult for acid aerosols, since there is no standard definition or measurement method for that pollutant, ambient concentrations and human exposures to acid aerosols are not well characterized, and the deposition and fate of acid aerosols in the respiratory tract are not well understood and are complicated by the neutralization of inhaled acid by ammonia in the human airways (EPA 1988a). Research on these issues is in progress, including research in our laboratories as well as others on the respiratory deposition issue (Bowes et al. 1989).

Human clinical studies have not demonstrated any effects of acid aerosols in the range of known ambient levels (which can apparently range up to about 40-50 $\mu g/m^3$ during episodic conditions). The studies do show that, at higher levels, asthmatics who are exercising or breathing through a mouthpiece (a problem discussed above) are more sensitive to sulfuric acid than are normal subjects. Even in such asthmatics, however, only modest bronchoconstriction has been found at concentrations of 400-1000 $\mu g/m^3$ (Utell et al. 1983) and either no effects or minimal reactions have been found at concentrations of around 100 $\mu g/m^3$ (Utell et al. 1983; Linn et al. 1986; Horstman et al. 1986; Koenig et al. 1983, 1985, 1989). In a recent report Hackney et al. (1989) concluded that "[t]he aggregate results suggest that only mild, if any, short-term respiratory irritant effects are likely at acid concentrations attained in ambient pollution." Such responses, like the responses of exercising asthmatics to SO_2, are not indicative of a threat to health. Moreover, "[e]xposures to combinations of sulfates with other pollutant gases, most notably ozone and SO_2, have not demonstrated any evidence of synergistic or interactive effects with endpoints that have been measured in human exposure studies" (EPA 1988a, p. 5-40).

Epidemiological data on the human health effects of acid aerosols in the real world are extremely limited at present. Clearly, much more research is needed before any conclusions can

be drawn from such data as to the potential health effects of acid aerosols.

A recent symposium (Environ Health Perspectives, Volume 79, Feb. 1989) has surveyed the subject admirably. Review of the papers from that symposium, as well as the advice given to EPA by its independent scientific advisory committee, CASAC (EPA 1988b, 1988c), makes quite clear that there are many unresolved questions about human exposure to and the possible health effects of acid aerosols. Until we have better information on those issues I do not think that there is a justifiable scientific basis for concluding that acid aerosols at current ambient levels are adversely affecting the public health.

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13 April 1989

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